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(Original Official Organ U. S. Vet. Med. Ass'n.)

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AVIAN PATHOLOGY NUMBER

CONTENTS

Editorial 543
Applications for Membership 546
Coming Veterinary Meetings 548
Papers:
Fowl Pest in the United States—J. R. Mohler 549
Fowl Pest—E. L. Stubbs 560
Infectious Bronchitis of Fowls—J. R. Beach 570
Avian Hemorrhagic Septicemia (Fowl Cholera)—J. W. Patton 581
Studies of Salmonella Pullora—Russell E. Rebrassier 603
Neuritis in Chickens—L. P. Doyle 622
Studies in Transmission of Bacillary White Diarrhea in Incubators—W. R. Hinshaw, C. W. Upp and J. M. Moore 631
B. Aertrycke Infection in Canary Birds and Parrots—F. R. Beaudette 642
B. Aertrycke as the Etiological Agent in a Disease Affecting Squabs—F. R. Beaudette 644
Plan for Handling Avian Tuberculosis—T. E. Munce 653
Clinical and Case Reports:
An Outbreak of Fowl Cholera—A. K. Gomez 657
Abstracts 664
Army Veterinary Service 669
Communications 670
Association Meetings 671
Miscellaneous 674
Necrology 676
Personals 679

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Vol. LXVIII, N. S. Vol. 21 **February, 1926** **No. 5**

POULTRY DISEASES AGAIN

Within a period of less than a year, we are presenting the second issue of the JOURNAL in which all of the papers, as well as much of the other material, are devoted to avian diseases. The first poultry disease number, published in May, 1925, attracted a great deal of attention, both within and without the profession. In a great many instances, this issue was an eye-opener for quite a number of men, not veterinarians, but closely associated with our great poultry industry.

Many veterinarians are putting forth an honest endeavor to be of service to the owners of poultry. On the other hand, it is a rather sad commentary to state that others apparently care nothing for poultry practice. It is true that veterinarians who completed their college training more than five years ago received very little instruction on the subject of poultry diseases in the regular veterinary course. During recent years, however, many of our veterinary colleges have been awake to the situation, and poultry diseases are now given the attention which their importance merits.

New conditions bring new problems, and just as veterinarians have been called upon to solve new problems in connection with horses, cattle, sheep, swine, and pet animals, they will also be called upon to solve the new problems in connection with poultry,

which are bound to develop with a changing poultry husbandry, bringing new methods of breeding, feeding, housing, and marketing. Every veterinarian located in a rural community should be just as close a student of poultry as of all other animals.

Unless those of our clients who are owners of poultry can be made to feel that veterinarians regard poultry practice just as much a part of their work as any other, we cannot hope to expect these owners of poultry to go to the veterinarian when they are in trouble. On the other hand, they will be much more likely to go to the drug store, the feed store, or the grocery store, and purchase some proprietary remedy, or they will rely upon the ever-increasing army of itinerant chicken doctors, who conduct farm-to-farm campaigns for the purpose of selling their nostrums.

In some states, every encouragement has been given practicing veterinarians by their state institutions, either through the extension service or otherwise, to become better posted on the subject of poultry diseases. A number of state veterinary associations have featured poultry diseases on the programs of recent meetings. Short courses have been offered, and numerous opportunities have been placed at the disposal of veterinarians in these states to enlarge their knowledge of poultry diseases. There does not seem to be much hope for those veterinarians who show no inclination to avail themselves of these splendid opportunities for enlarging their field of usefulness. Fortunately, they represent a group that is growing numerically smaller as time passes.

EXECUTIVE BOARD ELECTION

This is the year for the election of an Executive Board member from District Number 6, consisting of the following states: California, Nevada, Utah, Colorado, Kansas, Missouri, Arkansas, Oklahoma, Louisiana, Texas, New Mexico, and Arizona. Mexico and Central America are also embraced in this district.

Ballots on which nominations may be made, will be mailed on or about February 15, 1926, to all members in this district who are in good standing. Members whose dues for 1926 are not paid, are not in good standing and will not be entitled to vote until these dues are paid.

As there are approximately 700 members in District Number 6, there should be some lively voting. The nominating polls will be open for sixty days, which really should be long enough

for anyone to make up his mind as to how he wants to vote. As we have said in connection with previous elections, here is a chance to stay at home and vote too.

PENNSYLVANIA SCORES AGAIN

In this issue, as a contribution to our knowledge of poultry diseases, announcement is made of the first comprehensive plan for the control of avian tuberculosis to come to our notice. For several years veterinarians have been confronted with the fact that avian tuberculosis must be considered in any plan for the control of tuberculosis in other animals, particularly swine. Considerable research work has been done, surveys made, data accumulated and information disseminated, but no one has come forth recommending a definite plan for eradicating the disease from our farm flocks, until the announcement of the so-called Pennsylvania plan for handling avian tuberculosis, published in full in this number of the JOURNAL.

The Keystone State has always been a leader in animal disease control. The first state live stock sanitary board was organized in Pennsylvania over thirty years ago. The first law requiring the tuberculin testing of cattle was placed on the statute books in Pennsylvania about that time. Pennsylvania was the first state to buy and establish a farm where infectious and contagious diseases could be scientifically studied under actual farm conditions, through the assistance of a completely equipped laboratory maintained in conjunction with the farm. More recently the so-called Pennsylvania plan for the prevention, repression and eradication of bovine infectious abortion has received the nation-wide attention of veterinarians. And now, from the same source, we are given what appears to be a safe, sound and sensible plan for eradicating avian tuberculosis. Congratulations, Pennsylvania.

WHY NOT COMPARATIVE MEDICINE?

In opening the recent Conference of Veterinarians conducted by the School of Veterinary Medicine of the University of Pennsylvania, Dr. Josiah T. Penniman, president of the University, paid a glowing tribute to the veterinary profession and spoke very optimistically of the future of veterinary medicine, referring to the new fields and opportunities which have opened up to the graduate veterinarian. He referred especially to the

need of and opportunities for properly equipped veterinarians in public health and research work.

Dr. Penniman suggested, as have others, that because of the broadening of the field for veterinary service, which is no longer limited to equines but embraces cattle, swine, sheep, poultry and small animals, also research, regulatory and public health work, the word "veterinarian," which is a derivation of the Latin word "veterinarius," meaning "of or pertaining to beasts of burden or draft," such as the horse, no longer expresses adequately the scope and importance of the so-called "veterinary" profession in its true relation to economics and public health. President Penniman suggested the advisability of changing "veterinarian" to a more suitable name.

His is a constructive and timely suggestion. As to whose specific duty it is to decide the question we do not claim to know. Perhaps our "veterinary" schools or the American Veterinary Medical Association, or both, are the proper institutions to pass upon the suggestion. At any rate, Dr. Penniman has made a splendid point and it should receive full and impartial discussion. To that end the writer offers as a suggestion that the name "School of Veterinary Medicine" at the various institutions be changed to "School of Comparative Medicine;" also that the degree of "Doctor of Comparative Medicine" be conferred upon those graduating from such a course, instead of the degree of "Doctor of Veterinary Medicine."

T. E. M.

APPLICATIONS FOR MEMBERSHIP

(See October, 1925, JOURNAL)

FIRST LISTING

ALLERTON, FRED R. Hamlin, Kans.
D. V. M., Kansas State Agricultural College, 1925
Vouchers: Chas. W. Bower, R. C. Moore and B. W. Conrad.

ALSTON, JOSEPH THOMAS Tupelo, Miss.
M. D. C., Chicago Veterinary College, 1911
Vouchers: R. H. Mohlenhoff and Wm. P. Ferguson.

BANISTER, ROBERT MARTIN Sumrer, Miss.
D. V. M., Indiana Veterinary College, 1918
Vouchers: R. H. Mohlenhoff and Wm. P. Ferguson.

BARGER, EDGAR H. University Farm, Davis, Calif.
D. V. M., Kansas State Agricultural College, 1921
Vouchers: John L. Tyler and F. M. Hayes.

BLASER, CHARLES D. 711 Genesee St., Buffalo, N. Y.
V. S., Ontario Veterinary College, 1907
Vouchers: C. R. Perkins and F. E. McClelland.

CRAWFORD, WILLIAM R. College Ave., College Park, Md.
D. V. M., Cornell University, 1924
Vouchers: E. M. Pickens and Herbert L. Gilman.

EMMERSON, M. A. Iowa State College, Ames, Iowa.
D. V. M., Iowa State College, 1925
Vouchers: W. F. Guard and H. E. Bemis.

GATES, CHARLES H. Liberty, Ind.
D. V. M., Indiana Veterinary College, 1921
Vouchers: L. C. Henderson and E. V. Alexander.

HAGYARD, EDWARD W. 144 E. Short St., Lexington, Ky.
V. S., Ontario Veterinary College, 1888.
Vouchers: W. W. Dimock and H. Preston Hoskins.

KILLPACK, M. LLOYD R. F. D. No. 3, Box 183, Sandy, Utah.
D. V. S., Colorado Agricultural College, 1924
Vouchers: H. J. Frederick and Geo. H. Glover.

KINNEY, JAMES ROSER Netcong, N. J.
V. M. D., University of Pennsylvania, 1925
Vouchers: John W. Patton and Bruce Blair.

KREIDLER, HENRY E. Spartansburg, Pa.
V. M. D., University of Pennsylvania, 1921
Vouchers: J. H. McNeil and John T. McGrann.

MCINTOSH, G. E. 309 Central Ave., Louisville, Ky.
D. V. M., United States College of Veterinary Surgeons, 1923
Vouchers: F. M. Kearns and E. Calldemeier.

MCPHAIL, JAMES A. Manistique, Mich.
D. V. S., Grand Rapids Veterinary College, 1904
Vouchers: F. K. Hansen and E. D. Shevalier.

NEWMAN, W. P. Perham, Minn.
M. D. C., Chicago Veterinary College, 1911
Vouchers: J. H. Newman and Chas. E. Cotton.

SARDE, ROBERT M. Camden, Del.
V. M. D., University of Pennsylvania, 1913
Vouchers: C. C. Palmer and C. J. Marshall.

SEBASTIAN, GEORGE FINK Rehrersburg, Pa.
V. M. D., University of Pennsylvania, 1925
Vouchers: E. T. Booth and John W. Adams.

SNYDER, ORRIN W. Liberty, Ind.
D. V. S., American Veterinary College, 1884
Vouchers: L. C. Henderson and E. V. Alexander.

SMITH, FRASER A. 144 E. Short St., Lexington, Ky.
V. S., Ontario Veterinary College, 1900
Vouchers: W. W. Dimock and H. Preston Hoskins.

STEWART, ROBERT HYRAM Belzoni, Miss.
B. S., Mississippi A. & M. College, 1917
D. V. M., Alabama Polytechnic Institute, 1922
Vouchers: O. M. Norton and R. H. Mohlenhoff.

WAGHORN, T. G. Brockville, Ont.
V. S., Ontario Veterinary College, 1895
Vouchers: A. W. Younghusband and F. H. S. Lowrey.

WINKLER, J. A. Prospect & Elm Sts., Newport, Ky.
D. V. M., Cincinnati Veterinary College, 1918
Vouchers: Harry Gieskemeyer and H. Preston Hoskins.

Applications Pending

SECOND LISTING

Jones, Eugene Calhoun, 8670 Santa Monica Blvd., Los Angeles, Calif.
Manning, M. M., Bagley, Iowa.
Robertson, Duncan M., 7 Hartley St., Brockville, Ont.
Stauffer, L. R., 3800 Grand River Ave., Detroit, Mich.

Reinstated

Balthaser, H. B., 4907 Market St., Philadelphia, Pa.
Beavers, J. A., Canton, Miss.
Botkin, G. E., Mooreland, Ind.
Bouscein, G. L., Spring Valley, Minn.

Boyd, G. A., 4234 Euclid St., San Diego, Calif.
Boyd, Ralph H., 446 E. 10th St., Indianapolis, Ind.
Burcham, D. H., 9 So. 2nd St., Kansas City, Kans.
Busher, W. M., Novato, Calif.
Camuti, Louis J., 56 W. 66th St., New York, N. Y.
Chalmers, D. J., Grant, Iowa.
Ditto, John K., Eminence, Ky.
Gandy, M. H., 1810 Cherokee Ave., Baton Rouge, La.
Heth, C. M., Richland Center, Wis.
McCloskey, A. J., Elmendorf Farm, Lexington, Ky.
McKim, O. E., 425 Boston Post Road, Port Chester, N. Y.
Mann, Bernard, 1536 No. 8th St., Philadelphia, Pa.
Rafnel, R. V., Box 725, Jackson, Miss.
Ross, Harry H., 402 10th St., Brandon, Man.
Scarborough, W. T., 129 E. Davie St., Raleigh, N. C.
Trippeer, H. A., Snake River Route, Lewiston, Idaho.

COMING VETERINARY MEETINGS

Alabama Veterinary Medical Association and Short Course for Practitioners. Auburn, Ala. Feb. 1-2-3-4-5-6, 1926. Dr. C. A. Cary, Secretary, Auburn, Ala.

Iowa Veterinary Association. Savery Hotel, Des Moines, Iowa, and Iowa State College, Ames, Iowa. Feb. 2-3-4-5, 1926. Dr. E. R. Steel, Secretary, Grundy Center, Iowa.

Connecticut Veterinary Medical Association. Hotel Garde, New Haven, Conn. Feb. 3, 1926. Dr. Geo. E. Corwin, Secretary, 11 Warrenton Ave., Hartford Conn.

New York City, Veterinary Medical Association of. Academy of Medicine, 17 W. 43rd St., New York, N. Y. Feb. 3, 1926. Dr. C. G. Rohrer, Secretary, 40 W. 61st St., New York, N. Y.

Hudson Valley Veterinary Medical Society. Albany, N. Y. Feb. 10, 1926.

Conestoga Veterinary Club. Lancaster, Pa. Feb. 11, 1926. Dr. Henry S. Weber, Secretary, 27 E. Liberty St., Lancaster, Pa.

Florida State Veterinary Medical Association. Tampa, Fla. Feb. 12-13, 1926. Dr. A. L. Shealey, Secretary, University of Florida, Gainesville, Fla.

Illinois Veterinary Conference, University of. Urbana, Ill. Feb. 16-17, 1926. Dr. Robert Graham, University of Illinois, Urbana, Ill.

Keystone Veterinary Medical Association. Philadelphia, Pa. Feb. 24, 1926. Dr. G. A. Dick, Secretary, 39th St. & Woodland Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. American House, Boston, Mass. Feb. 24, 1926. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

FOWL PEST IN THE UNITED STATES*

By J. R. MOHLER, *Washington, D. C.*

Chief, U. S. Bureau of Animal Industry

INTRODUCTION

Longfellow's saying, "Disasters come not singly," aptly expressed our sentiments last fall when, following the strenuous campaigns against foot-and-mouth disease in California and Texas, we were confronted with an outbreak of European fowl pest, a disease which until that time had never appeared in this country. When and where the first outbreak occurred is not definitely known. It seems probable, however, that the disease made its appearance in New York City and New Jersey and on a few farms in the vicinity of those places as early as September, 1924.

Dr. A. Silkman, Chief Veterinarian, New York City Health Department, telephoned the Bureau the latter part of October, stating that poultry dealers in his city were experiencing heavy losses among chickens shipped in from the Central West, and wished to know if we had any information on the subject. He stated that their laboratory had been unable to help them, and promised to send us some sick birds for examination, but for some reason or other this was not done. On November 13, Dr. V. A. Moore, of Cornell University, brought to our attention a disease in poultry on Long Island and at Liberty, New York, which, as he expressed it, "seems to be fowl plague. * * * "The lesions resemble those described for fowl plague." Later, on December 11, we received a second letter from Dr. Moore, in which he definitely stated: "I believe the disease we have here is identical with it (fowl pest)."

During this period, reports reached the Bureau that many birds were dying in fattening establishments in the Central West. As soon as these reports were received, we detailed employes to investigate conditions in those sections where the heaviest losses were occurring. It was soon determined that two diseases, somewhat similar but not identical, were causing these losses, resulting in much confusion in the region where deaths were first reported. Diseased birds were secured for

* Presented at the sixty-second annual meeting of the American Veterinary Medical Association, Portland, Oregon, July 21-24, 1925.

laboratory examination and, early in December, we were able to announce that one of the affections had been diagnosed as fowl pest, the other as infectious bronchitis. The latter, and not fowl pest, seems from our present knowledge to have been responsible for most of the losses last fall in the fattening establishments in the Central West, the non-filtrability of its infective agent clearly differentiating it from fowl pest.

Nature of the Disease

Fowl pest is an acute infectious disease of fowls caused by a filtrable virus. It was first described in 1878, by Perroncito, but first differentiated from fowl cholera in 1880, by Rivolta and Delprato. Finding its origin in northern Italy, the disease has since spread into the Tyrol, Germany, Belgium, France, Argentina, and Brazil.

While numerous investigators (Foa, Demel, Abba, Belfanti, Zenoni, in 1899) were claiming the discovery of the causative germ, Centanni and Savonuzzi, in 1900, were apparently first in recognizing the ultramicroscopic character of the virus. They too first demonstrated its filtrability.

Kleine and Möller compared the disease with rabies, calling attention to its effect upon nerve centers of geese. Rosenthal (1905) called attention to the presence of bodies resembling the Negri body of rabies. Hoare suggests that the disease may have existed unrecognized for a considerable time among poultry, due to the similarity which it has to other septicemic diseases, particularly fowl cholera.

The infective principle of fowl pest has been found in the blood (with strong affinity for the red corpuscles), brain, nerve tissue, nasal secretions, inflammatory exudates, bile and feces of affected fowls. A small piece of liver from the carcass of an infected chicken will produce death if fed to another chicken. The disease may be transmitted experimentally by the subcutaneous injection of as little as 0.000.001 cc of virulent blood. The filtrates, though virulent, possess a much lower degree of potency than the unfiltered virus, and are correspondingly easier to destroy.

Natural infection occurs by the association of healthy birds with diseased ones, or by placing healthy birds in an environment recently infected. Likewise, migratory birds may become infected and carry the infection to distant points, before they themselves succumb and, by dying, render a new center of

infection liable.[1] Rodents (themselves not susceptible) may become mechanical carriers of infection in their quest for food.

The transportation of diseased fowls proved to be one of the most prolific means of dissemination in this American outbreak of the disease. The improper disposal of infected carcasses in particular proved a serious handicap to its control. A particular instance illustrates this point. In one of the states, several fowl pest carcasses were thrown into an empty coal car from a poultry car on an adjoining track. Susbequently, this coal car was transported to a distant state, where unsuspecting railroad hands threw out the dead hens along the siding. Chickens foraging in this vicinity were not long in acquiring the infection, and a new center of fowl pest was thus established.

According to Hoare, chickens, turkeys, geese, ducks, pheasants, peafowls, guinea fowls, pigeons, parrots, blackbirds, and canaries are susceptible to the disease. Hutyra and Marek, however, definitely except wild waterfowls and pigeons, particularly with regard to natural infection. Mammals are not susceptible to the infection.

In the work done by various American investigators, it was generally agreed that the strain of fowl pest virus recently prevalent in parts of this country possessed a particular virulence for chickens and to a lesser degree for turkeys, and that ducks were not affected by natural exposure. Experiment ducks, geese, and pigeons, at the Bureau of Animal Industry, exposed either by feeding or intramuscular inoculation, failed to show any ill effects, while chickens and turkeys were consistently susceptible. Krumweide, Gerber, and Provost, however, report the death of a pigeon from the subcutaneous injection of 0.00,000,001 cc of virus; a duck, 0.5 cc; a goose, 0.1 cc; and a guinea hen, 0.0,000,001 cc. Various mammals, including two (*Macacus*) monkeys, were inoculated by these workers, with negative results.

The incubation period of experimental infection is from thirty-six hours to two or three days. Natural exposure may require even five to seven days.

Symptoms

The symptoms of the disease are not particularly characteristic, and might be mistaken for fowl cholera or other acute infections. The bird shows a rapidly progressing droopiness and depression, with loss of appetite and fever. The comb,

wattles, eyelids, and earlobes take on a pale, purplish hue, and become increasingly edematous as the course of the disease advances. The edema may even descend into the neck and under the breast, and is usually found on autopsy to be of a clear, serous nature, straw-colored, or possibly flecked with fibrin. The feathers are ruffled and soiled.

In the advanced stage, the bird appears to be irresistibly sleepy and may even sink into an uninterrupted coma, which, however, may culminate in a violent death-convulsion. During the disease, a few cases manifested a greenish-yellow diarrhea, difficult to distinguish from that of fowl cholera. Some also had a clear, viscid lacrimation and nasal discharge, the eyes and nostrils becoming clogged and pasted, even in early stages of the disease. It is thought that some of the respiratory symptoms frequently noted in connection with the American outbreak might have been attributed to complication with the very widespread infectious bronchitis. This hypothesis seems borne out by the significant silence of most European writers as to respiratory symptoms. Freese goes so far as to discredit the pathological significance of the mucous accumulations found in the nasal cavity and throat, believing that these are normal secretions that could not be expelled. None of the experiment fowls which died of fowl pest at the Bureau of Animal Industry showed any symptoms or lesions which could have been confused with those of infectious bronchitis. Andrien and Badano (Argentine Republic) describe a nervous type of the disease characterized by ataxia, with movement of the head in a circle.

Lesions

Upon opening the carcass, many cases show a more or less extensive gelatinous edema under the skin, or even in the heart sac or body cavity.

The classical lesion of European fowl pest, however, is the hemorrhagic condition of the mucous lining of the proventriculus or glandular stomach. These hemorrhages may be petechial or ecchymotic, and may involve the glands in particular, or may indiscriminately splotch the mucosa.

Next in importance are ulcerative or hemorrhagic lesions found in the underlying surface of the gizzard, or muscular stomach, after the cuticle has been removed.

Various other organs and parts are likely to show hemorrhagic spots of varying proportions, as, for instance, the subcostal

region of the chest wall, the sternum, heart fat, gizzard fat, and mesenteric fat. Congestion may also be in evidence in the liver, kidneys, spleen, and ovary. Localized hemorrhagic patches are sometimes visible in the mucosa of the duodenum. In well-marked cases, longitudinal, hemorrhagic striations have been observed on the serous surface of the rectal wall.

Differential Diagnosis

In view of the close clinical resemblance which exists between avian hemorrhagic septicemia (fowl cholera) and fowl pest, it has been necessary to adopt routine precautions in the differentiation of these conditions. Virulent material from a suspected case of fowl pest is diluted in normal saline solution and passed through a bacteria-retaining filter (Berkefeld or Mandler). Cultures are made from the heart-blood and organs of the carcass to determine the presence of visible pathogens. Cultures of the virus are made before and after filtering, as a control on the reliability of the filter. The absence of bacteria in the filtrate, taken together with its ability to produce the symptoms and lesions above described, are regarded as laboratory proof of its identity with the virus of fowl pest.

Fowl cholera may also be readily detected by the inoculation of rabbits, as these are highly susceptible to the cholera organisms, but are naturally immune to the virus of fowl pest.

Immunology

For obvious reasons, medical treatment has been found unsatisfactory. According to a personal letter, dated March 14, 1925, received from Dr. C. M. Haring, while at Zurich, Switzerland, a "triple vaccine" is being distributed by Professor Terni throughout northern Italy for immunization of poultry against fowl pest, fowl cholera, and avian diphtheria. The fowl pest vaccine (which alone concerns us here) consists of virulent brain and medullary substance ground in carbolized saline solution (1 part tissue, 50 parts solution) and allowed to stand for 10 days. No data are available as to its efficacy.

Maggiora and Valenti, and also Kraus and Loewy, immunized adult geese against the virus of geese by inoculation of a gallinaceous strain of the virus. The geese after being hyperimmunized in this manner, yielded a serum which would protect chickens if given in large injections.

Attempts to immunize chickens by the injection of virus attentuated by exposure to chloroform vapor were unsuccess-

ful in our Bureau laboratory. No further experiments in the immunology of this disease in America are recorded.

Control Measures

On December 11, 1924, New York City placed an embargo against shipments of poultry originating in North Dakota, South Dakota, Nebraska, Kansas, Missouri, Iowa, Illinois, and Indiana. The next day, the state authorities of New York quarantined against shipments from these same states, and in a few days so many states had issued orders restricting or absolutely prohibiting poultry shipments that it was practically impossible to move live poultry interstate to the great consuming centers of the East. Prices broke badly, and producers and distributors suffered heavy losses. Dressing establishments, especially in the East, were forced to discontinue operations, and thousands of employes were thrown out of work.

No money was available for poultry disease work in the field, but, realizing the seriousness of the situation, immediate steps were taken to secure an appropriation. President L. B. Kilbourne, of the National Poultry, Butter and Egg Association, President S. S. Knight, of the Petaluma Poultry Growers Association, and others interested in the poultry industry, hastened to Washington. Following a conference in my office on December 17, the matter was brought to the attention of Congress, which promptly authorized the use of $100,000 from the emergency appropriation for foot-and-mouth disease work. The bill making this amount available, was signed by the President on December 24. However, anticipating favorable action, we had, on December 22, issued B. A. I. Order 291, which prohibited the interstate shipment of live chickens, turkeys, and geese infected with or directly exposed to European fowl pest or similar contagious disease of poultry, and which required the cleaning and disinfection, under Bureau supervision, of cars, coops, troughs, and other accessories that had been used in the interstate transportation of poultry infected with such diseases.

At that time, it was known that fowl pest existed at several of the large eastern terminal markets and that cars and coops at such markets had contained infected birds. As it was impossible to determine which cars and coops were infectious, drastic action was considered necessary to prevent widespread dissemination of the disease. Accordingly, as a preliminary measure to the enforcement of B. A. I. Order 291, our inspectors-

in-charge in twenty-seven States, i. e., Minnesota, South Dakota, Nebraska, Kansas, Oklahoma, Missouri, Kentucky, Tennessee, West Virginia, Maryland, the District of Columbia, and all states to the north and east thereof were instructed, on December 23, to require the cleaning and disinfection of all assembling and feeding plants, cars, coops, crates, troughs, and other accessories that had been used in the handling of interstate shipments of poultry during the thirty days immediately preceding that date. These instructions, sent to about 140 Bureau stations, immediately brought into service in this campaign against fowl pest more than 500 of our employes. Letters were also sent to officials in charge of live stock sanitary control work in the states named, requesting their aid in this undertaking. The response from such officials was most gratifying, and the states in every instance took care of a large share of this work.

General Cleaning and Disinfection

Our reason for requiring this general cleaning and disinfection was not because the disease had been found in all these states, but because it was impossible to tell how widely cars and coops that had been used in the handling of diseased poultry had been distributed. Severe winter weather prevailed while this work was being carried on, temperatures below zero being not uncommon in some of the states. This was the first time any federal disease-restrictions had ever been placed on the poultry industry, and its members naturally were unfamiliar with the manner in which it was necessary to accomplish cleaning and disinfection to comply with Bureau requirements. In spite of these obstacles, the state and federal employes engaged on this work, which involved the cleaning and disinfection of 2718 plants, 8140 cars, 352,525 coops, and 124,997 pieces of miscellaneous equipment, prosecuted the work with such vigor and skill that it was practically completed by March 1, 1925.

The early completion of this general disinfection was due in no small degree to the hearty cooperation which we received from all branches of the poultry industry. As an example of such cooperation, I desire to mention a conference at Chicago, Illinois, on January 16 and 17, which was called by the National Poultry, Butter and Egg Association to discuss the poultry disease situation. This meeting, which was largely attended by representatives of the poultry industry and by federal and state officials, formulated recommendations with respect to

more uniform methods of handling the campaign against fowl pest, and did much to establish closer and more cordial relations between the agencies interested in this work. In addition to this precautionary disinfection, Bureau employes were detailed to investigate all reports of suspected outbreaks of fowl pest and also to make surveys to determine infected areas.

Extent of the Outbreak

Outbreaks of fowl pest occurred in the states of Pennsylvania, New York, New Jersey, Connecticut, Indiana, Michigan, West Virginia, Missouri, and Illinois. The disease existed in the first four of these states before eradication work was commenced. Most of the infection was found in the markets in Philadelphia, New York City, Jersey City, and three or four cities in Connecticut. In all four of these states, however, outbreaks occurred on a number of farms, the infection usually being traceable to birds purchased at markets in the cities referred to, or to coops returned from such cities. After the general disinfection was well under way, and it was no longer possible to move diseased or exposed birds, few outbreaks occurred in these states. New Jersey and Connecticut have been free from the disease since the latter part of January. No outbreak has occurred in New York since March 15. (Since this was written, the disease has been diagnosed on Long Island, where all but 18 in a flock of 900 chickens died. No spread from this point has thus far been reported.) There was, however, an outbreak in Pennsylvania, involving a small flock of chickens as late as May 2. In Indiana, the disease was diagnosed about February 1. Outbreaks occurred at three loading stations and on six farms. The disease was also found in February in Michigan and Missouri. In the former, outbreaks were confined to markets in the city of Detroit and to one feeding station outside that city. In the latter, infection was found only on the premises of a poultry firm in St. Louis. The outbreaks in these three states, apparently traceable to infected cars which were removed from eastern terminals before we knew the disease existed in this country, were easily controlled. In West Virginia, only one diseased flock was found, the outbreak occurring on February 10. In Illinois, the disease made its appearance about April 3, in some small flocks in a suburb of Chicago. By the latter part of the month, this outbreak was suppressed.

This was the extent of the disease in this country, with the exception of infection in several cars which were returned from eastern terminals a few days before Order 291 was issued. These cars were intercepted on their way back to the points at which they had been originally loaded, and all the birds which they contained were properly disposed of under the supervision of Bureau or state inspectors. In no instance, so far as we were able to ascertain, was there a spread of infection from such cars to country flocks. As an example of the virulence of fowl pest, attention is invited to a death loss, in five or six days, of 3000 birds out of 3300 in one of these cars.

Methods somewhat similar to those employed in combating foot-and-mouth disease were used in eradicating fowl pest. Inspectors were accoutered in rubber outfits, affected flocks were slaughtered and, together with the birds that had died, were burned or deeply buried. Infected premises were thoroughly cleaned and disinfected under official supervision, and subsequently test birds were introduced on such premises before restocking was permitted. In one or two states, the owners were reimbursed for the fowls which were destroyed. The Bureau, however, did not participate in the payment of this indemnity. As with foot-and-mouth disease, we found evidence in at least one of these outbreaks which indicated that birds may recover and still be carriers of the infection. This shows the importance of disposing of all birds in an affected flock in such a way as absolutely to preclude exposure from such a source.

Standardized Control Measures Desirable

By means of press releases, important features of the work were discussed from time to time. In one of these, the Bureau referred to complaints it had received that large quantities of poultry had been contaminated with the odor from coal-tar disinfectants and suggested this trouble might be avoided by the use of hot lye solution or a 4% solution of formaldehyde. In another, it was explained that the Bureau had no control over state embargoes, but was endeavoring to have the states coordinate and standardize, so far as practicable, their regulations on this subject. In still another release, we stressed the importance of sanitation as an important factor in combating poultry diseases. As indicated in a statement which we gave to the press on March 16, 1925, tubes of fowl pest virus were brought from France in September, 1923, by an investigator connected

with a large eastern institution, who had been working on the subject of filtrable viruses for some time. While there is a possibility that the disease may have been introduced through some other channel, the fact remains that this is the only definitely known introduction of the virus.

It is most difficult to estimate, with any degree of accuracy, the losses occasioned by this outbreak of fowl pest. My reason for this statement is that losses caused by infectious bronchitis and other less virulent diseases of poultry were, in many instances, attributed to fowl pest. Available data indicate, however, that direct losses from fowl pest were not less than $1,000,000, and probably considerably greater. As was the case in California last year, the drastic embargoes imposed by some of the states caused losses far exceeding those arising from the death of infected birds, and the enforcement of reasonable federal and state regulations.

While present conditions are excellent, I feel, in view of the extent of the outbreak, that more time should elapse before we can be positive that the disease has been completely stamped out. In the meantime, the Bureau is exercising the utmost vigilance, and is prepared to combat the disease promptly and effectively in the event it should recur.

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DISCUSSION

DR. F. J. BOLENDER: How long were the premises kept under quarantine?

DR. MOHLER: In this outbreak of fowl pest, which occurred in the winter time, the time elapsing after disinfection and before removing the quarantine was all the way from two weeks to thirty days. When the quarantine was

removed the owner was permitted to restock gradually, the first birds added being used as test birds to ascertain if any infection remained. In this connection I would like to say that it would be easier to eradicate half a dozen outbreaks of fowl pest than one outbreak of foot-and-mouth disease. Rinderpest and fowl pest are similar in their high death rate. In these diseases the situation is largely cleared up by the rapid death of the birds or the animals on infected farms. In the fowl pest outbreak, after disinfection was accomplished, we have allowed restocking of the premises to begin within two weeks after the final disinfection.

ANNUAL CONFERENCE OF KANSAS VETERINARIANS

The fifth annual conference of Kansas veterinarians will be held at Manhattan, in the veterinary buildings of the Kansas State Agricultural College, February 10-11, 1925. Dr. W. L. Williams, of Ithaca, N. Y., will deliver an illustrated lecture on "The Prognosis of Genital Diseases, Based on Clinical History and Lesions," the morning of the first day. In the afternoon he will give a laboratory demonstration, assisted by Drs. Frick and Bullard, of K. S. A. C.

Other extra-state contributors will be Dr. L. Van Es, of the University of Nebraska; Dr. E. R. Steel, of Grundy Center, Iowa; Drs. J. C. Flynn, and A. T. Kinsley, of Kansas City, Missouri. Dr. N. L. Townsend, of the U. S. Bureau of Animal Industry, will speak on the progress of tuberculosis eradication in Kansas. Dean Dykstra and his efficient staff will round out the program with presentations of their particular specialties.

Three "good old heads" of Missouri who were among those present at the first special course for graduate veterinarians at the University of Missouri, Dec. 28-31, 1925: (left to right) Dr. D. B. Morgan, Neosho; Dr. W. B. Welsh, Marshall and Dr. J. W. Connaway, Columbia.

FOWL PEST*

By E. L. STUBBS

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During the fall and winter of 1924-1925 a serious disease affecting poultry was found in the United States which had never before been reported in this country. This disease, fowl pest or fowl plague, seems to have first caused severe losses among live poultry in the New York City market. Dr. J. R. Mohler,[1] chief of the federal Bureau of Animal Industry, reported its existence in this country in December, 1924. Dr. E. L. Brunett[2] reported, in the January, 1925, number of the JOURNAL of the American Veterinary Medical Association, "The Occurrence of a Disease of Chickens in New York State, Caused by a Filterable Virus." The writer[3] of the present article, in the University of Pennsylvania Veterinary Extension Quarterly, on January 3, 1925, reported its presence in Pennsylvania. In the May, 1925, issue of the JOURNAL it was also reported from New Jersey,[4] Indiana,[5] Illinois,[6] and Michigan.[7] Dr. L. D. Krohn,[8] of the New York City Department of Health, reported in the May, 1925, issue of the JOURNAL, "A Study of the Recent Outbreak of Fowl Disease in New York City." He stated that "unusual losses were occurring as early as the latter part of August." Since that time this disease was found in other poultry markets, particularly Jersey City and Philadelphia, and was spread to many native flocks where severe losses occurred.

Fowl pest was most prevalent at Thanksgiving and Christmas time. With the placing of embargoes, quarantines and restrictions on shipping poultry, together with the cessation of traffic in live fowl following the holiday season, there was a marked diminution in the cases of fowl pest reported. The vigorous measures instituted in the way of restriction on shipping and quarantine, as well as the cleaning up and disinfecting of the poultry markets, no doubt had a very beneficial effect on the control of this disease, as well as other poultry diseases. It is very fortunate that this disease was recognized and measures instituted for its control, because it is perhaps the most fatal disease of fowl and, should it make inroads into our poultry

*Presented at the sixty-second annual meeting of the American Veterinary Medical Association, Portland, Oregon, July 21-24, 1925.

flocks, would produce severe losses that would be felt not only by the producer but also by the consumer. The dangerous character of this disease warranted any radical measures taken for its control and if it is successfully eradicated that feat can be recorded as a worthy accomplishment of the American people.

Information regarding this disease must be sought for in articles written by European investigators. It seems first to have been investigated and described in Italy, about 1878. It was found to be a disease different from fowl cholera, in Italy, in 1880, and Centanni and Savonuzzi in 1901 found that the cause was capable of passing through a porcelain filter. It raged severely in northern Italy in 1894 and from there spread into the Tyrol and Germany, later into Belgium and France. It was spread broadcast over Germany in 1901 by the Brunswick Fowl Exposition and has often been called the Brunswick fowl disease.

Etiology

Fowl pest or fowl plague is an acute, highly fatal disease of fowl and related species. It is caused by a filtrable virus. Very small amounts of this virus, or materials which contain it, are highly infective. The virus is known to be present in the blood, in the nasal and oral secretions, in the intestinal and urinary excretions, in the mucous and serous exudates. It is present in the nervous system and probably in all tissues and tissue juices of the body. Filtrates of such materials even in high dilution also contain the virus.

This virus, like other viruses, is easily killed. Exposure to direct sunlight or to a temparature of 70° C. for a few minutes renders it inactive. Its activity is destroyed quickly by the common disinfectants. Desiccation is not rapidly fatal to it and under proper conditions it will retain its virulence for long periods. Blood placed in glass tubes, sealed and kept in a dark place, retains its activity for three months. In Pennsylvania we have kept washed red blood cells, from an affected chicken, in a test-tube unsealed for three months and, upon mixing this material with salt solution and injecting it into chickens we produced the disease. Filtrates retain their virulence with less uniformity.

Pathogenicity

Fowl pest attacks fowl and related species. Chickens and turkeys are most susceptible and most often found affected. Pigeons are quite resistant, as also are water fowl, ducks and geese. Such fowl are infected with difficulty, even artificially,

and are rarely found naturally affected. Mammals are immune. It is believed that this disease is not dangerous to man, since mammals are immune and there are no recorded cases in humans.

With few exceptions, chickens can be infected with ease. A few seem to be immune. We still have one of the first chickens brought to our laboratory last fall, from an affected flock, and it is immune. Chickens are easily infected by the injection of quantities as small as one millionth of a cubic centimeter of blood or filtrate. They may be infected by subcutaneous, intravenous, intramuscular or intraperitoneal injection. Infection also succeeds easily when such materials are fed. Injuries to the skin, with introduction of the virus, or instillation into the eye, produce the disease. After such infection the birds die in 36 to 72 hours as a rule and may or may not show symptoms. Pigeons are quite resistant, even when injected. In Philadelphia, at the Pennsylvania Bureau of Animal Industry Laboratory, last winter we injected 16 pigeons with virulent fowl pest virus and in only two instances did any die. These showed no symptoms and only slight lesions. However, when a portion of the brain of one of them was injected into a chicken, it died with characteristic lesions. In several of our most virulent outbreaks ducks and geese were associated with the dying chickens but in no case were any found affected. On one premise, where 300 chickens died in about 10 days, there were associated 30 geese and 250 ducks, but none of those water fowl died. We have also injected a number of rabbits, guinea pigs and white mice with 1 cc of virulent virus, but in no case was sickness or death produced.

Outbreaks of fowl pest were investigated on two premises where baby chicks were being raised. None of the chicks became affected. These chicks were not in close association with the affected flock.

Three chicks, three to eight weeks old, were injected subcutaneously with fowl pest virus. One chick of the same lot was left as a control. The three injected chicks were dead on the third morning. The control chick which was in the same cage was dead on the seventh day after the three other chicks were injected, and four days after the three injected chicks had died.

Two older chicks, about 12 weeks old, were injected subcutaneously with virus. Two chicks of the same lot were placed in the same cage as the controls. The two injected chicks were dead the second day following injection. One of the other chicks which had not been injected was dead, five days after the two

chicks were injected which was three days after they had died in the same cage. The other control chick in the same cage remained healthy.

These chicks did not show marked lesions of fowl pest. All did show slight hemorrhages in the proventriculus. The two control chicks, one of the younger lot and one of the older lot, both of which died after natural infection by contact, did show slightly greater hemorrhages in the proventiculus.

The transmission of this disease has not been definitely worked out. It is thought that the nasal, oral and fecal materials contaminate the food and water, thus accounting for its spread. However, it is a very interesting fact that chickens, placed in containers in which deaths have occurred, do not always become affected. We have repeatedly used cages, in which chickens have died of fowl pest, for other chickens, without even cleaning or disinfecting and the birds placed in them did not become infected. The birds later proved to be susceptible by artificial injection. Without doubt the diseased bird is by far the most dangerous factor in the spread of this disease.

Symptoms and Course

Fowl pest, as we have found it in Pennsylvania, is a very acute disease and symptoms may not be noticed. When symptoms are observed dullness and inappetence appear first. The hens stop laying, the feathers are ruffled and the birds become weak and staggery. Occasionally the head is extended and there is a gasping for breath. Some show a catarrhal or mucous discharge from the nostrils and mouth which may be streaked with blood. There may be a diarrhea. Fever may be quite marked and we have recorded temperatures as high as 111°F. The head is at first red, later becoming cyanotic, and may be swollen. The wattles and ear lobes may show marked edema. The eyes may be swollen, in some cases entirely closed. The swollen heads, as we have found them in Pennsylvania, give a striking clinical picture.

The course is quite rapid, chickens living only a few hours, rarely more than 24 hours after showing symptoms. In our work, chickens usually died in 36 to 72 hours after artificial inoculation. Death takes place after a short struggle and quite often the chicken is found dead on its back.

Postmortem

The postmortem changes in fowl pest are marked. The following is a description of lesions we have found in the outbreak in Pennsylvania:

Rigor mortis occurs rapidly and is complete. Cyanosis of the head is noticed. Edema may be present in the wattles, in the ear lobes, about the eyes or in the loose subcutaneous tissue. The edematous fluid is clear and straw-colored. On removing the skin all blood-vessels are found engorged with blood. The meat is red and frequently hemorrhages may be noticed in the muscles on the breast. When the breast is removed, very distinct petechial hemorrhages may be found on the inner surface of the breast bone. These are quite characteristic and appear as though blood had been sprayed on with an atomizer.

The characteristic tissue alteration in fowl pest is hemorrhage. These vary from the smallest possible petechial hemorrhages to ecchymoses. When the hemorrhages are small and widely scattered they may be missed easily. When they are grouped, as in the proventriculus or over the abdominal fat, they are marked in appearance. These hemorrhages may occur in any tissue. The most characteristic lesion is the hemorrhagic alteration in the proventiculus. The hemorrhages can be seen after the whitish mucus is washed off the mucous membrane. Such hemorrhages are usually in the form of ecchymoses, which may occur between the conical elevations or secreting glands of this stomach. They are perhaps more noticeable when they occur as bright red blotches on the mucous membrane, where it becomes smooth, to enter the gizzard. Petechiae or ecchymoses are also seen in the gizzard, after the rough membrane or cuticle has been removed. The intestine frequently shows hemorrhagic changes, especially in the duodenum. These hemorrhages, in the form of petechiae or ecchymoses on the serous or mucous coat, are accompanied by a catarrhal enteritis. In fat birds, petechial hemorrhages are particularly noticeable in the abdominal fat, especially over the proventriculus, gizzard and on the fat of the mesentery. The functional ovary shows highly engorged blood-vessels.

Occasionally a serous or sero-fibrinous exudate may be found in the pericardium. Petechial hemorrhages occur frequently on the pericardium and especially over the base of the heart. Petechiae and ecchymoses also occur on the mucous membrane of the mouth, about the tongue and in the esophagus. In some

cases the trachea is quite reddened. The lungs may be found congested and petechiae may occur on the pleura.

Autopsies of 106 chickens affected with fowl pest from natural infection, occurring in outbreaks in Pennsylvania, show the following conditions: symptomatically, cyanosis of the head, 75 per cent; swollen wattles, 29 per cent; swollen ear lobes, 22 per cent; and swollen heads, 19 per cent. Pathologically, hemorrhages in the mucous membrane of the proventriculus, 91 per cent; hemorrhagic changes in the gizzard, 56 per cent; hemorrhages in the abdominal fat, 55 per cent; hemorrhages on the heart, 26 per cent; hemorrhages in the intestine, 10 per cent; hemorrhages on the inner surface of the breast bone, 10 per cent; and hemorrhages on the mucous membrane of the mouth, about 10 per cent. The lesions varied in intensity as well as in distribution.

Treatment

No satisfactory treatment is known for fowl pest. Prevention, control and eradication are the measures which must be used in handling this disease.

On December 16, 1924, the Pennsylvania Bureau of Animal Industry sent a circular letter to all veterinarians in Pennsylvania notifying them of the existence of fowl pest in Pennsylvania, giving a description of the symptoms and lesions, and asking their cooperation in handling it. In that letter the following recommendations were made:

"Poultrymen should be warned against adding new fowl to their flocks. Where additions must be made the added fowl, regardless of the source, should be isolated for a period of at least one week. In any case all coops, containers and carriers should be cleaned and disinfected or burned. Measures must be taken to destroy and limit the infection. Sick fowl should be destroyed and all carcasses should be immediately burned or properly buried. Frequent diligent cleaning of the infected premises, coops, crates, other containers and cars, followed by thorough disinfection is essential. The dangerous and common method of spreading the disease is the intermingling of fowl, exposure to infected premises, coops, crates, other containers and carriers."

A letter was also sent to all county agents, transportaion companies, newspapers, and other poultry agencies, notifying them of the presence of fowl pest and asking their cooperation in reporting and handling it.

All reported cases of fowl pest in Pennsylvania were investigated by the Bureau of Animal Industry, the fowl quarantined and the premises disinfected before being released from quarantine. The Federal Bureau of Animal Industry and municipal departments of health cooperated with the Pennsylvania Bureau of Animal Industry in cleaning up and disinfecting the poultry markets which were clearly the source of infection in Pennsylvania. The infected fowl and the infected coop or crate have been generally considered the greatest source of danger in the spread of this disease. The poultry handlers had been subjected to practically no restraint in their business prior to the fowl pest outbreak and, in the beginning, considerable difficulty was experienced with them. However, many of them saw the benefits of cleaning and disinfecting and say they will continue that practice.

Specimens of diseased fowl were brought to the Laboratory of the Pennsylvania Bureau of Animal Industry from natural outbreaks in twenty-one native flocks. In fifteen of these there were clear histories of the introduction of the disease by the addition of new fowl. The most of these cases occurred around Thanksgiving and Christmas time. Several of the outbreaks resulted from the purchase of a holiday fowl, either a chicken or turkey, on the poultry market. Such fowl were in contact with the flock for as short a time as an hour or a few days, with the consequent loss of large numbers and the entire flock in two cases.

The following is a typical example: The day before Thanksgiving, a man purchased two live turkeys at the Philadelphia poultry market. They were not killed for Thanksgiving. The morning after Thanksgiving one turkey was dead, the other one was sick and was killed. The following day one chicken died. From then on, six or eight chickens were found dead each day until every one in the flock of 98 died. After about 70 of these chickens had died the owner called a practicing veterinarian, who brought dead fowl from this premise to our Laboratory. They showed typical lesions of fowl pest. Blood was passed through a Berkfeld filter and the filtrate produced the disease with typical symptoms and characteristic lesions.

When we first encountered fowl pest we suspected fowl cholera, because of the rapid onset, high mortality, prostration of individuals and, most of all, because of the hemorrhagic lesions found. Repeated bacteriological examinations were entirely negative, as well as rabbit and guinea pig inoculations. After

several autopsies we found that we were encountering a uniform set of hemorrhagic lesions, particularly in the proventriculus and gizzard. A search of the literature was made, and fowl pest most closely resembled the disease we had found. Further work satisfied us that we were dealing with that disease.

Differential Diagnosis

In a consideration of the differential diagnosis, fowl cholera must be placed first because each of these two diseases presents similar symptoms and lesions. Symptoms in common are: rapid onset, high mortality, prostration, cyanosis and diarrhea, and, above all, lesions of a hemorrhagic character. Mortality in fowl pest is usually regular, while in fowl cholera it is more likely to be irregular. In cases of natural outbreaks, where chickens are associated with water fowl, as ducks or geese, in fowl pest these do not become affected, while in fowl cholera ducks and geese are highly susceptible and show fowl cholera to a marked degree.

In fowl cholera, where several autopsies are made, usually some present livers with areas of focal degeneration, showing as whitish-yellow points scattered over the liver. Such changes have not been found in fowl pest. Hemorrhages are more likely to be scattered throughout the body, while in fowl cholera they are more often confined to the heart and intestine.

Where recourse is had to laboratory examination in fowl pest, cultural examination is negative, inoculations into rabbits, mice and pigeons are negative, while the filtrates will produce the disease. In cholera cultural examinatoin is positive, inoculation into rabbits, mice and pigeons produces death and filtrates do not cause the disease. Fowl typhoid is a more chronic disease, although differentiation may require the isolation of typhoid-like organisms. Apoplectiform septicemia and sleeping sickness cause symptoms and lesions similar to fowl pest, but can be differentiated by the demonstration of a streptococcus in the blood stream. Phosphorus poisoning produces lesions in the proventriculus of a hemorrhagic nature and may be confusing. Such hemorrhages are much more deeply seated and are accompanied by erosions of the mucous membrane, which is not the case in fowl pest. Besides, the odor of phosphorus is of help in such a differentiation.

Simultaneously with the fowl pest outbreak there occurred what seemed to be an independent disease but which was confused with it. This has been named infectious bronchitis and is

characterized by an inflammation of the trachea and bronchi. The most of the trouble from this disease has been experienced in the large feeding stations. If affects and causes losses in birds of all ages, but is most severe in the young ones. The symptoms usually noticed are an extension of the neck, an opening of the beak with a gasping for breath. There is inappetence and considerable distress. The head becomes cyanotic and many die suddenly. Postmortem lesions are confined to the respiratory tract.

Fowl pest is a definite disease entity. It is caused by a filtrable virus which attacks fowl and related species, producing a disease plague-like in character.

Fowl pest can be diagnosed when a plague-like disease is encountered in fowl, showing the symptoms of an infectious disease, with cyanosis and edema of the head, and hemorrhages in the proventriculus, gizzard and abdominal fat. In cases of doubt, negative bacteriological examinations, negative results from inoculations into mammals, and filtrates producing the disease with typical symptoms and characteristic lesions remove the question of doubt.

The occurrence of fowl pest has stimulated considerable thought regarding transmissible diseases of poultry. It has also drawn the attention of the public to the value of the veterinary profession in the prevention, control and eradication of infectious diseases as well as to the service the veterinarian can render in handling poultry diseases.

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DISCUSSION

DR. R. R. DYKSTRA: At the time that fowl pest was more or less prevalent in the eastern states an embargo was placed on the shipment of poultry from the middle western states, and I wondered many times why that embargo was placed; whether there was any evidence at all that seemd to make the eastern people think that the poultry disease that was in the West might be fowl pest.

DR. STUBBS: I am not able to give an opinion on how great the necessity was for placing embargoes. I think the original embargo on the West was placed by New York City. I can speak only from our position in Pennsylvania. When we first diagnosed this disease, the restrictions applied to every state;

we did not discriminate between the West or South or any other section. We required that poultry from all the states, coming into Pennsylvania, be inspected and found free from disease. Shortly after that, many states sent information to our state that fowl pest did not exist in those states, and our restrictions were removed then from all the states except those in which it had been definitely diagnosed. From conversation with men in the East it does seem as though many fowls did die in the New York City market, unmistakably from fowl pest, and that is given as the reason for placing the embargoes.

CHAIRMAN STEEL: I think about that time we had another epizootic in the Central West, a condition resembling roup, and probably there was some confusion regarding that.

DR. J. R. BEACH: In California we were naturally very much interested and worried when we heard that fowl pest had been found in the eastern states, and there was a temporary embargo placed by California against shipment of poultry from the Middle West at the same time as by the eastern states. That was maintained only until we could get some definite information regarding how widespread fowl pest was. One reason for placing that embargo immediately was the loss, in a carload of live poultry from Nebraska, of over sixty per cent, en route. This, as we determined from an examination, was not due to fowl pest, but to infectious bronchitis. It has been very definitely determined that there is no relationship whatsoever between fowl pest and infectious bronchitis.

I made a tour of inspection in the Middle West and East, to satisfy myself and our people in California that there was no relationship between these two diseases, and I got the impression quite definitely that it was this infectious bronchitis which was responsible for the embargo being placed by the city and state of New York against the middle western states. I may say in regard to fowl pest that I found that the best source of information was Dr. Stubbs. I visited all of the laboratories in all the sections where fowl pest occurred and was being studied. I might just as well have confined my visit to Pennsylvania and Dr. Stubbs. That is where I got all the information that I needed.

FARM RADIO SCHOOL AT M. S. C.

The Michigan State College is conducting the second Farm Radio School, from Station WKAR. Programs are broadcast five evenings each week from 7:15 to 8:00 p. m., eastern standard time. Each Tuesday evening the Animal Husbandry, Poultry, and Veterinary Medicine Course is broadcast.

During January the following lectures were given by members of the Veterinary Division:

Jan. 12. Veterinary Education—Dr. Ward Giltner.
Jan. 19. The Development of the Chick Embryo—Dr. F. W. Chamberlain.
Jan. 26. The Digestive Tubes of Farm Animals—Dr. H. E. Johnson.

The following program has been arranged for February and March:

Feb. 2. Meat Inspection—Dr. E. T. Hallman.
Feb. 9. The Udder of the Cow in Health and in Disease—Dr. L. B. Sholl.
Feb. 16. The Care of the Pleasure Horse—Dr. J. P. Hutton.
Feb. 23. Veterinary Practice with Small Animals—Dr. E. K. Sales.
Mar. 2. The Glands that Control Personality—Prof. B. B. Roseboom.
Mar. 9. Methods and Uses of Antiseptics and Disinfectants—Dr. H. L. Morency.
Mar. 16. Poultry Diseases—Prof. W. L. Mallmann.
Mar. 23. Vaccination in Veterinary Practice—Dr. I. F. Huddleson.
Mar. 30. Clean Milk—Safe Milk—Prof. G. L. Ruehle.

INFECTIOUS BRONCHITIS OF FOWLS*

By J. R. BEACH

Division of Veterinary Science, Agricultural Experiment Station, University of California, Berkeley, Cal.

The occurrence of unusually high mortality among live poultry shipped from the Middle West to eastern markets led, in December, 1924, to the placing of an embargo by the state and city of New York against poultry from seven middle western states. The presence of fowl pest in New York and adjoining states was detected at about the same time and, consequently, it was stated that the losses in the poultry originating in the Middle West were from this cause. Investigation, however, showed conclusively that fowl plague was not present in any middle western state and that the losses that occurred in market poultry from those states were from a disease which, if not new, had not previously been extensively studied.

This disease has been variously designated as infectious bronchitis, infectious tracheitis, tracheo-laryngitis, chicken "flu" and Canadian "flu." Because of its prevalence among fowls in feeding stations and during shipment, the disease has been likened to "shipping fever" of live stock. We have accepted the name "infectious bronchitis," used by Hinshaw,[1] in view of his finding that the disease starts as a typical bronchitis.

The length of time poultry of this country have suffered from the disease is not definitely known. Operators and employes of feeding stations claim that it has been present for many years, but losses caused by it have not been serious previous to the fall of 1924. In some poultry periodicals, the statement is made that it was introduced into this country by show birds from Canada in 1921. The first authentic report of its occurrence in the United States, however, appears to be that of May and Tittsler,[2] regarding an outbreak of tracheo-laryngitis in a Rhode Island flock in November, 1923. The first known occurrence of the disease in California was in November, 1924, in a flock of pullets. During the following few months, numerous other outbreaks occurred in poultry flocks in widely separated districts. Quite heavy losses were also sustained among fowls in fattening stations. These were chiefly among live fowls shipped from the Middle

*Presented at the sixty-second annual meeting of the American Veterinary Medical Association, Portland, Oregon, July 21-24, 1925.

West (Nebraska), but also to some extent in fowls obtained from California flocks.

Infectious bronchitis has occurred to a greater extent among fowls in feeding stations and in poultry cars during shipment to market than among poultry on the farms. Hinshaw[3] and Eriksen[4] state that losses were heavier among young birds than among old ones, although fowls of all ages were affected. In outbreaks investigated by the writer, the age of fowls in the flocks varied from seven months to two years. It was noted, however, that the mortality was less and continued over a shorter period in the flock of two-year-old hens than in the flock of seven-months-old pullets.

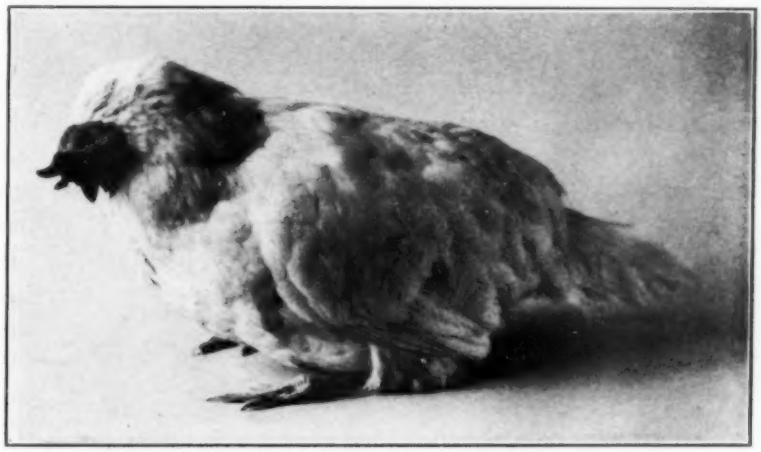

FIG. 1. An advanced case of infectious bronchitis. Attitude during expiration.

The predominant symptom of the disease is "gasping for breath." The affected fowl assumes a sitting position, with the neck drawn in, the beak pointed downward and the eyes closed (fig. 1). At each inhalation, the head is thrown forward and upward, with the beak opened and the intake of air is accompanied by a loud wheezing sound (fig. 2). Spasmodic exhalation or coughing is frequent and often results in the expulsion from the trachea of a mass of clotted blood. Examination may reveal the superior larynx to be nearly filled with a caseous mass, while in other cases, in the live bird, the cause of the respiratory difficulty is not visible. Collections of mucous or caseous exudate are frequently found within the eyelids. This causes the eyelids to become glued together and, in case both eyes are affected, makes it impossible for the fowl to see. There

is usually a discharge from the nostrils. In many cases, this results in the infraorbital sinuses becoming filled and distended. This condition is indistinguishable from the commonly-occurring roup.

Hinshaw[3] states that cases which survive for a week develop a typical avian diphtheria. In our experience, however, the fowls exhibiting respiratory symptoms only either died within two or three days or made complete recovery. In fact, our observations led us to believe that recovery was not infrequent when there was no serious involvement of either the eyes or nasal sinuses.

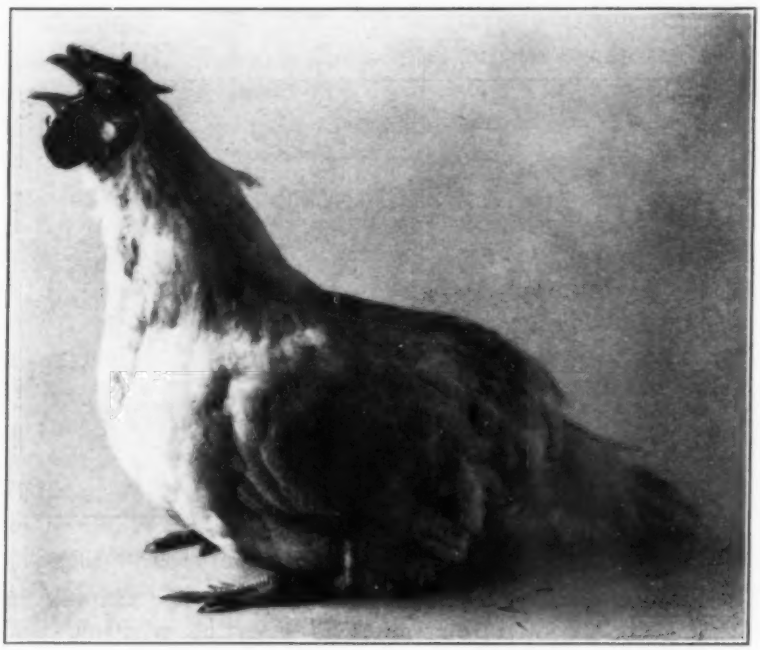

FIG. 2. Same fowl as in fig. 1. Attitude during inspiration.

The period of incubation and course of the disease are very rapid. In feeding stations, many fowls died within six or seven days after exposure. In fowls that were artificially infected, symptoms developed and death occurred in from three to seven days after inoculation.

Hinshaw[1] reports that in feeding stations the mortality ranged from 20 per cent to 30 per cent of all birds in the feeding batteries during a fattening period of from five to eleven days.

In regard to losses among live poultry in transit from collecting or feeding stations to distant markets, the writer has definite information regarding only those that occurred in shipments from

Nebraska to Los Angeles, San Francisco and Oakland. Approximately 60 per cent of one carload of over four thousand fowls died en route and many more were sick on arrival at their destination. In all other cases, the loss per carload during transit was from one hundred to two hundred birds. This number is but little in excess of the normally-expected mortality. Additional birds became affected and died, however, after removal from the cars to the dealers' establishments. The extent of this additional mortality is not known.

The rapidity of development and losses caused by infectious bronchitis on poultry farms is shown in the following account of three outbreaks:

Outbreak No. 1: The first outbreak occurred in a flock of 2250 pullets, seven months old. These birds were in excellent condition and had just begun to lay well. The owner had observed no evidence of disease previous to finding four dead one morning, in one pen of 1100 birds. Four days later, the number of deaths had mounted to eighty. The losses continued to increase until the eighth day, when seventy-five birds died, and then rapidly subsided. Within three weeks after the first death in this pen, sickness and death had ceased. The disease then appeared in another pen and followed an almost identical course. From a total of 2250 birds, 510 died from infectious bronchitis. Production dropped from two hundred eggs per day to thirty-five in each pen, within a week after the onset of the disease. The owner estimated his loss through the death of birds and the decrease of egg production to have been in excess of fifteen hundred dollars.

Outbreak No. 2: This was in a flock of 1100 hens, two years old. On February 6, 1925, droopiness and difficult respiration in a few birds were noticed. Between February 6 and 10, the mortality was four to five birds a day and then increased to from eighteen to twenty-two birds a day until February 14. On this date about 10 per cent of the flock were visibly sick but there were only four additional deaths. The total mortality in this flock of 1100 hens was 110. The egg production fell from 62 per cent, on February 6, to 10 per cent, on February 14. The production had returned to normal in four weeks after the outbreak started.

Outbreak No. 3: The third outbreak occurred in a flock of approximately 2000 one-year-old hens, and caused a loss of 150 birds during a period of one month. By the end of this time,

the disease had run its course and the flock was again healthy. The distribution of mortality and the effect of the disease on the egg production in the flock were not obtained.

Postmortem Findings

On autopsy, all organs are apparently normal with the exception of the superior larynx, the trachea, the inferior larynx and the bronchi. The mucosae of these organs are usually found inflamed and their lumens wholly or partially filled with mucus and clotted blood. In some cases, probably those of several days' duration, the mucus in the superior larynx has become caseated. Occasionally caseous mucus is found extending the entire length of the trachea. In some birds, the amount of clotted blood present exceeds that of mucus. The collection of mucus is sometimes confined to the lower portion of the trachea and inferior larynx, the upper portion of the trachea and the superior larynx being entirely free.

The degree of inflammation of the mucous membrane varies from slight to an extent such as to impart a bright scarlet color to the membrane. Hinshaw[3] states that the exudate also extends into the smallest bronchi. This may have been the case in the autopsies made by us, but our examinations were not searching enough to reveal it. May and Tittsler[1] also apparently overlooked this condition, if it was present, in the birds they examined. The lungs appear normal except for small areas of congestion. Death in many cases appears to be due entirely to asphyxiation, caused by the filling of the superior or inferior larynx with caseated mucus or clotted blood.

Etiology

The etiology of the disease is still undetermined. Hinshaw[3] points out that birds in feeding stations become affected following exposure to cold during hauling in open trucks or in poultry cars. He also noted that the disease appeared on Kansas farms following shipment of fowls to the farms from poultry shows. The disease on the farms subsided when the fair and show season was ended. He further observed that the losses at the feeding stations were heaviest among birds that were in poor condition as a result of inadequate diet or intestinal parasitism. He concludes, therefore, that exposure and undernourishment are important factors in the causation of the disease.

May and Tittsler[2] also state that three of four outbreaks observed by them appeared to have been brought on by exposure

to cold. No history of such an occurrence could be obtained in connection with any of the flocks affected in California. Neither could it be said that these flocks were not properly fed or that the fowls were in poor condition. On the contrary, the disease in these flocks seemed to attack principally the best birds. A similar observation was made by Hinshaw[4] in some outbreaks on the farms.

While it is probably true that exposure and malnutrition may be factors in the production of infectious bronchitis in fowls, the results obtained by all who have studied the disease definitely show that it can be readily transmitted from sick to healthy fowls that have not been exposed to cold or other unfavorable environment. This has been accomplished both by confining healthy birds with sick ones and by inoculating healthy birds with tracheal exudate from affected birds.

Gwatkin[5] claims to have produced both typical chicken-pox and avian diphtheria lesions by inoculating healthy cockerels with an emulsion of tracheal exudate after filtration through a Berkefeld filter. Cultural tests showed the filtrate to be free from bacteria. The fowl from which the material for inoculation was obtained showed no lesions except bloody mucous exudate in the trachea. He considers, therefore, that the disease is caused by the filtrable virus of chicken-pox and that it is an acute form of chicken-pox or avian diphtheria.

May and Tittsler[1] transmitted the disease by the introduction, by means of a swab, into the mouth and trachea of healthy birds, of material from the trachea of dead birds. Sickness and death were produced in three of six fowls inoculated in this manner.

Hinshaw[2] also succeeded in infecting healthy birds by intratracheal inoculation with exudate from the trachea of affected birds. In one case the typical respiratory symptoms and lesions developed after intra-abdominal inoculation. Extensive bacteriological studies of the exudate were also made by this investigator. Out of sixty-four fowls examined, the fowl cholera organism was isolated twenty-one times and a diplococcus thirty-seven times. The colon organism was encountered in a few cases. In the exudate from some birds, no bacteria at all were found. Experiments to determine the relationship of these organisms to the production of infectious bronchitis of fowls have not been carried out.

In our studies of the etiology of infectious bronchitis, we have used material from sick or dead fowls from four California flocks

in which outbreaks of the disease occurred and from two cars of live market poultry shipped from Nebraska to Oakland or San Francisco. The lesions found in all of these birds were the same as previously described. With the exception of a few birds in which the eyes and nasal sinuses were involved, lesions were confined to the larynx and trachea. Cultures were made from the heart-blood and liver of each bird. *Bact. avisepticum* was isolated from one bird which had been dead about thirty-six hours when it was examined. In all other cases, however, no growth of bacteria was obtained.

Transmission Experiments

Healthy fowls and rabbits were inoculated in various ways with saline emulsions of exudate from the trachea of these naturally-infected birds and of other birds which became diseased from inoculation. A summary of the results of the inoculations follows:

Tracheal exudate was introduced, by means of a swab, into the trachea of fourteen healthy cockerels. Ten of them developed the characteristic respiratory symptoms and died in from four to six days after inoculation. Three became sick but recovered. One bird was not affected.

Nine cockerels were given an intra-abdominal inoculation. Exudate collected in the larynx and trachea of three of them, causing their death. Two that were inoculated with exudate from Nebraska birds died from infection with *Bact. avisepticum*. Four birds were not harmed by the inoculation.

Ten cockerels were given an intravenous injection of a saline emulsion of exudate from the trachea. Four died within thirty-six hours after inoculation. Pure cultures of *Bact. avisepticum* were obtained from the heart and liver of each. The source of the exudate with which these birds were inoculated was the fowls from Nebraska. The remaining six cockerels were not affected.

The comb of each of twenty-six cockerels was scarified with a needle dipped in exudate from trachea of diseased birds. Definite lesions of chicken-pox developed on the combs of six birds on the seventh day after inoculation. One of these also developed characteristic respiratory symptoms. One of the birds that was inoculated with exudate from the Nebraska birds died from fowl cholera. Nineteen of the twenty-six birds were unaffected. The chicken-pox lesions were confined to birds inoculated with tracheal exudate from fowls from three of the four California flocks.

Twelve of seventeen rabbits that were given intra-abdominal injections of saline emulsion of tracheal exudate died within thirty-six hours after inoculation. Pure cultures of *Bact. avisepticum* were secured from the heart-blood and liver of each. The other five rabbits remained healthy. In every instance, inoculation with exudate from the naturally-infected fowls caused death of the rabbits. The ones that survived were from those inoculated with material from birds in which disease was produced by artificial infection.

Cockerels were inoculated with cultures of *Bact. avisepticum* isolated from the rabbits. One of three inoculated intraperitoneally died from acute fowl cholera. The others were not affected. All of eight cockerels given intratracheal inoculations remained healthy.

A portion of the saline emulsion of exudate from the larynx and trachea of affected fowls was filtered through a Mandler filter candle. Cultural tests showed the filtrate to be free from living bacteria. Cockerels were inoculated with the filtrate as follows: four, intratracheally; seven, intra-abdominally; eight by scarification of the comb, and seven, intravenously. Peritoneal injections were also given to eight rabbits. None of the chickens or rabbits was in any way affected by the inoculations.

Chicken-pox Virus Present

Inoculations with saline emulsions of exudate before filtration had showed that some of them contained the filtrable virus of chicken-pox and it was expected that this filtrable virus would also be present in the filtrate of those emulsions. The failure of chicken-pox lesions to develop on susceptible cockerels inoculated with the filtrate indicated that this was not the case. It seemed possible, therefore, that the filter used might be too fine to permit the passage of the virus. In order to determine the correctness of this idea, an emulsion of chicken-pox virus was filtered through the same candle. Cockerels inoculated by scarification of the comb with a needle dipped in this emulsion before filtration developed chicken-pox lesions after the usual incubation period for the disease. Cockerels inoculated in the same manner with the filtrate, however, remained healthy. These results indicated that the virus had been removed by the filtration.

In a further attempt to determine if chicken-pox virus is a factor in the production of infectious bronchitis of fowls, tracheal exudate from affected birds was ground in a mortar with 50

per cent glycerin solution containing 0.5 per cent phenol. This mixture was then stored in an ice-box until, by cultural methods, it was found to be sterile. In other experiments (unpublished) we have found that in mixtures of chicken-pox virus with 50 per cent glycerin solution containing 1 per cent phenol, the virus was still virulent after storage for a year and one-half.

Seventy days after the tracheal exudate-phenolized glycerin mixture was prepared, it was applied to a scarified surface of the comb of two cockerels and introduced into the trachea of two others.

Chicken-pox was produced in both birds that were inoculated by scarification of the comb. The lesions appeared on the seventh day.

Both birds inoculated by the intratracheal method showed slight dyspnea on the sixth day. A small amount of caseous exudate was visible in the larynx of each. A few days later, this condition had disappeared.

A third attempt to demonstrate relationship between chicken-pox and infectious bronchitis was made. In this, chicken-pox virus was introduced into the trachea of two cockerels. One of them remained healthy. The other showed difficulty in respiration on the ninth day and died on the sixteenth day. On autopsy, the superior larynx and the upper portion of the trachea, for a distance of 2 cm., were found to be plugged with a mass of caseous exudate. This condition was similar to that found in some of the fowls that died from infectious bronchitis.

From the foregoing, it is seen that the etiology of infectious bronchitis of fowls has not as yet been determined. The important points in this connection that have been brought out are:

First: Infectious bronchitis is readily transmitted to healthy fowls by the introduction into the trachea of material from the trachea of an affected fowl.

Second: Chicken-pox virus is sometimes present in the exudate in the larynx and trachea of diseased birds. The importance of this virus in the etiology of the disease, however, is not definitely determined.

Third: The fowl cholera organism and a diplococcus are commonly present in the exudate in the larynx and trachea of naturally-infected birds. It has not been shown, however, that either of these organisms is concerned in the production of infectious bronchitis.

Fourth: Lesions very similar to those of infectious bronchitis were produced in one of two fowls inoculated by the introduction of chicken-pox virus into the trachea.

Control Measures

The control of infectious bronchitis appears to have been a greater problem in the feeding stations than on the farms. In the former, after the disease appeared, it continued to cause losses in each succeeding lot of fowls that was brought in from the farms, until very stringent sanitary measures were enforced. On the farms, however, outbreaks ran their course in from two to four weeks. There have been no recurrences of the disease in any of the California flocks which have been affected.

The measures adopted for control of the disease in the feeding stations consist of daily cleaning and disinfection of the coops, feed troughs, and all other accessories, and careful search for diseased birds, which are at once removed from the batteries. Hinshaw[2] states that chlorin disinfectants have proved satisfactory. Coal-tar disinfectants are unsuitable for use in feeding stations and on shipping crates, because of the danger of the odors being absorbed by the birds and the flesh thereby being rendered unfit for food.

Treatment

A number of methods for the treatment of diseased birds have been used. These include Dakin's solution or argyrol, applied to the affected part; two drops of beechwood creosote in cod-liver oil, given daily; 3-grain doses of quinin sulphate, given three times daily; 20 minims of aromatic spirits of ammonia, every two hours; 5 cc doses of flavisol, subcutaneously; 20 to 30 grams of calcidin, in hot mash, daily; and avian mixed bacterins. Definite information seems to be lacking, however, regarding the actual value of any of these.

The discovery of a successful method of treating sick fowls, on account of the small value of an individual bird, is of relatively, small importance, however, unless the treatment is inexpensive does not require frequent repetition and is highly successful. Of greater importance is a successful method of prevention. We will probably have nothing more effective than sanitary measures for prevention and control of infectious bronchitis of fowls until the etiology of the disease is determined.

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[2]May, H. G., & Tittsler, R. P.: Tracheo-laryngitis in poultry. Jour. A. V. M. A., lxvii (1925), n. s. 20 (2), pp. 229-231.
[3]Hinshaw, W. R.: An epizootic of bronchitis among chickens. (To be published.)
[4]Eriksen, S.: The epizootic of bronchitis among chickens. Year-book Mo. State Poultry Board, 1924, pp. 48-52.
[5]Gwatkin, R.: Some notes on avian diphtheria. Rpt. Ont. Vet. Col., 1924, p. 54-61.

DISCUSSION

DR. L. R. VAWTER: Does this condition exist only in the winter months or does it occur along into the warmer weather?

DR. BEACH: In the Middle West I believe it started in August. The first we saw of it was in November, which is not very cold, in California. We did not see very much of it after the first of the year, when we had our coldest weather.

HORSE TALKS BY RADIO

The following radio addresses were given during January, under the auspices of the Horse Association of America.

Jan. 7. Station WJY, New York. "Modern Polo," by Louis E. Stoddard, Chairman of the United States Polo Association.
Jan. 9. Station WOAW, Omaha, Nebr. "Horseback Riding for Children," by W. G. Langworthy Taylor.
Jan. 12. Station KYW, Chicago. "Living Locomotives," by Wayne Dinsmore, Secretary, Horse Association of America.
Jan. 14. Station WJY, New York. "Horsemanship in Polo," by J. Watson Webb.
Jan. 21. Station WJZ, New York. "The Horse and Mule in National Defense," by Major General J. G. Harbard.

Eighty per cent of all meat food animals produced in the United States are marketed through the 68 public stock yards at which central markets are located.

Three of the main attractions at the first special course for graduate veterinarians at the University of Missouri, Dec. 28-31, 1925: (left to right) Dr. E. T. Hallman, of Michigan State College; Dr. John W. Adams, president of the American Veterinary Medical Association; Dr. F. R. Whipple, Peoria, Ill., small animal specialist.

AVIAN HEMORRHAGIC SEPTICEMIA
(Fowl Cholera)

By JOHN W. PATTON, *Amherst, Mass.*[1]

Department of Poultry Husbandry, Massachusetts Agricultural College

The domesticated poultry of this country, as well as many species of semi-domesticated and wild birds, are susceptible to several types of highly destructive, communicable diseases, which, in the aggregate, cause a loss to the raiser, amounting to many millions of dollars annually.

In our present-day scheme of a complete and well-rounded successful system of agriculture, we find poultry-farming occupying a prominent and popular position. With this increased popularity and interest, it is but reasonable to expect a demand from poultry raisers for some definite knowledge as to the treatment and prevention of one of their most discouraging, as well as most frequent, causes of failure, certainly one of the least understood factors pertaining to the practice of successful poultry husbandry, namely, poultry disease.

That this demand for information and relief is increasing is evidenced by the rapidly increasing service demanded from the agricultural colleges of the country—postmortem examinations, laboratory diagnosis, serological tests and general correspondence. This demand has increased to such proportions, in several states, that it requires the undivided attention of several investigators.

Of the many diseases of poultry, avian hemorrhagic septicemia is without doubt, one of the most serious, because of its wide distribution, its acute, highly contagious nature, its potential destructive ability and, at the present time, the lack of suitable measures for its treatment and control. Not unmindful that Pasteur's original experiments, upon which our present-day knowledge of immunity is based, were carried out with this organism and disease, nor the apparent success of biological treatment in the hands of other investigators, yet in the light of our present knowledge of the disease, much information is to be revealed before commending any preventive or curative measures beyond those of sanitation, isolation, and quarantine.

[1]Resigned. Received for publication, March 31, 1925.

Historical Resume

This dangerous fowl plague was described as early as 1850 by Benjamin,[1] and by Delafont and Renault,[2] in 1851. Hering,[3] in 1859, recognized its contagious nature and substantiated it experimentally. Perroncito,[4] in 1879, also Semner[5] described a diplococcus, which they repeatedly found in the blood of fowls dead of the disease, as the causative factor of the malady, which they considered fowl cholera. Later work by Hadley and Amison[6] (1911) shows that the disease was probably fowl typhoid. Toussaint[7] (1879) and Pasteur[8] (1880) were successful in the artificial cultivation of the organisms, thus enabling them to study their pathogenic characteristics. Pasteur carried out his basic experiments on attenuation of bacterial cultures and immunization with such cultures, with the bacillus of fowl cholera. Salmon[9] (1879-80) investigated an outbreak of disease amongst poultry in South Carolina, which he concluded was fowl cholera. Petri[10] (1884) isolated a small bipolar organism from an epizootic amongst fowl, geese, and ducks. Cornil and Toupet[11] (1888) studied an outbreak of what they called "cholera des canards" (goose septicemia).

Loir and Declaux[12] (1894) isolated an organism, *B. avium*, similar to the organism causing cholera. Moore [13,14] (1895) investigated a fatal disease in pigeons, associated with an outbreak of hog cholera, and later a similar one in fowls. Hadley considered this a fowl typhoid infection. Higgins[15] (1898) reported an outbreak of cholera in Canada. Curtis[16] (1900) investigated a fatal outbreak amongst geese. Theobald Smith isolated a bipolar-staining organism in pure culture, and concluded that the disease was goose septicemia. Ward[17] (1904) studied an outbreak in California. Hadley and Amison[6] (1911) concluded, after a biological study of eleven pathogenic microorganisms from cholera-like diseases of poultry, that genuine fowl cholera, identical with that studied by Toussaint and Pasteur, is enzootic in New England and the Middle Western states, and that its prevalence is increasing. They further concluded that there existed other cholera-like diseases of poultry, in which the cholera organism is absent.

The organisms causing these diseases usually belong to the paracolon, paratyphoid, and influenza groups. Besemer[18] (1917), in his studies of the fermentation reactions of the hemorrhagic septicemia group of organisms, added considerably to the litera-

ture regarding these organisms. Beach[19] (1922) concluded that a high protein diet might lower the resistance of fowls, referring further to the localization of the disease in ovaries, joints, and wattles. In the "plague" outbreak of the present winter, several avian pathologists, Hinshaw[20] and Pyle,[21] have isolated cholera, or analogous microorganisms, from outbreaks, supposedly plague, brought to their attention. There is little doubt that plague existed during this time, however, as careful laboratory technic also brought to light complications of *P. avicida*.

Distribution

Fowl cholera occurs extensively in Europe, with the exception of Great Britain, and in North and South America. In Germany (1903-10) the numer of cases fluctuated, with the greatest loss occurring in 1906, numbering 63,329 cases. In Austria, where compulsory reporting of the disease has been in existence since 1903, the disease was reported in the year 1907 in 12,250 chickens, 1946 ducks, 2329 geese, 166 turkeys, and 41 other fowls. Roumania (1911) reported 9028 cases. The disease appears to be widespread in Russia, Italy, Hungary, Sweden, France, Bulgaria, and India, as well as in the United States, general opinion to the contrary notwithstanding. Compulsory notification of the presence of the disease exists in Germany, Austria, and Hungary, where, upon its appearance, the infected premises or, as the case may be, localities and even townships are quarantined against traffic in fowls. The inland and foreign traffic, as well as dealers' premises, are subject to official supervision.

Etiology

Pasteurella avicida, an organism of the hemorrhagic septicemia group. Synonyms: *Pasteurella avium*, *B. cholerae*, *B. cholerae gallinarum*, *B. bipolaris septicus*, *B. avisepticus*, *Pasteurella* (*aviseptica*) *cholerae gallinarum*.

Morphology

Microscopically, the fowl pasteurella is a small, rod-shaped bacillus, 0.25 to 0.4 μ in diameter and 1-2 μ long, with rounded ends, non-motile, occurring singly, non-spore-forming, non-capsular. Involution forms are frequently seen in old laboratory cultures and less frequently in freshly isolated specimens.

Staining: The bipolar arrangement of the protoplasm can best be demonstrated by the use of either Giemsa's or Ziehl-Neelson's carbol-fuchsin stain, especially when viewed from

TABLE I—*Fermentation reactions of P. aviculda (various investigators)*

	Fructose	Galactose	Dextrose	Mannose	Sorbose	Arabinose	Xylose	Rhamnose	Lactose	Maltose	Saccharose	Raffinose	Dextrin	Inulin	Erythrite	Adonite	Dulcite	Mannite	Sorbite	Amylose	Glycerin	Salicin	Isodulcite	Amygdalin
Magnusson	+	+	+	+	−	−	−	−	+	−	+	−	−	−	−	−	−	+	+	−	−	○	○	○
Veurland	+	−	+	○	○	−	−	−	−	−	+	○	−	−	−	−	−	+	+	○	−	○	○	+
Schirop	+	Tr.	+	○	○	−	○	○	○	−	+	○	○	−	−	−	+	+	○	○	−	○	○	○
Bessemer	+	+	+	+	○	○	○	○	−	−	+	−	−	−	−	−	+	+	○	○	−	−	−	+
Hadley	+	+	+	+	○	−	+	○	−	−	+	−	−	−	−	−	−	+	○	○	−	−	−	+
Mack-Records	○	+	+	○	○	○	○	○	−	○	+	−	−	○	○	○	○	+	○	○	○	○	○	○
Jackley	+	+	+	○	○	○	○	○	−	−	+	○	−	−	○	○	−	+	○	○	−	−	○	○
Tunnicliff	+	+	+	+	−	± [1]	+	−	−	− [3]	+	Tr.[2]	−	−	−	−	±	+	+	−	−	−	−	+
Patton	+	+	+	+	○	+	○	−	−	− [3]	+	−	−	−	○	○	○	+	○	○	−	○	○	○

+ = Acid. − = No acid. Tr. = Trace. ○ = No test. ± = Variable.
[1]Usually positive. [2]Trace or negative. [3]If chemically pure.

freshly prepared blood-smears. The organism stains readily with the ordinary dyes and is Gram-negative.

Cultural characteristics: The organism is primarily an anaerobe. In serum broth and when freshly seeded it will grow in media deprived of oxygen. Aerobic cultures grow feebly between the temperatures of 20-25° C., the optimum being 35-39° C.

Character of growth: Agar (veal infusion). On a 1-2 per cent neutral, or slightly alkaline, pH 7.2, and at a temperature of 37.5° C., growth is fairly rapid. It is essential that the agar be freshly prepared, with sufficient water of condensation present, otherwise the growth will be scanty and difficult to transfer. The colonies after 24-48 hours of incubation appear as small, round, shiny and thin discs, about 2 mm. in diameter. Under the two-thirds objective, they appear coarsely granular with concentric circular markings, smoky-brown in color by direct transmitted and greyish by reflected light. Subsurface colonies appear lenticular, and the granular appearance appears more marked under the two-thirds objective than in the surface colonies. After 48 hours of growth, the colonies change from a translucent to a smoky-brown color with undulate border. Any water of condensation becomes turbid. After the first week of growth, the liquid tends to clear, leaving a characteristic viscous sediment.

Blood or glycerin agar. Growth upon this medium shows no characteristics distinguishable from those observed upon plain agar.

Gelatin. The colonies are slower to develop and smaller. Otherwise they resemble those observed upon agar.

Bouillon (alkaline pH 7.2). After 12-48 hours of incubation a slight clouding appears, clearing after a week or ten days of growth, with more or less deposition of a viscous sediment. A slight circular band is sometimes seen at the level of the surface of the fluid; no pellicle is formed. The reaction is alkaline, especially in old cultures.

Sugar-free bouillon. On this medium, growth is similar to that observed on alkaline.

Milk. No change is observed within ordinary observations.

Indol formation. Indol is produced more rapidly, however, in some strains than in others

Reduction of nitrates. Nitrates are reduced to nitrites.

Fermentation reactions. One per cent solutions of the desired carbohydrates in sugar-free, veal-infusion broth, pH 7.2, using

brom-thymol blue, give very satisfactory results. For general laboratory diagnosis, the four common sugars used and their reactions are as follows:

Dextrose	Lactose	Maltose	Saccharose
+ —	— —	— —	+ —

Source of Infection

This is most generally accomplished through the medium of the digestive tract, contaminated droppings, saliva of infected fowls, viscera or other parts of the carcass of birds dead of the disease, feeding and drinking utensils, and so forth. The disease may be introduced onto clean premises by recently purchased birds, or those returning from shows, contests, and the like. Pigeons, sparrows, and other free-flying birds should not be overlooked as a possible source of infection. External parasites, flies, fleas, ticks, and mites may have some bearing, as Hertel[22] demonstrated virulent bacilli in dermanyssus mites which he obtained from the bodies of fowls ill from the disease. Shilston,[23] writing of the disease from India and speaking of bovines and wild game, cites Dammann and Oppermann and their incrimination of biting flies (*Simulium ornata*). According to Gamaleia,[24] the intestinal canal of healthy pigeons contained fowl cholera bacilli. This could not be substantiated by the work of Ostertag and Ackerman[25] and Joest[26] in reference to chickens, whereas, Muller[27] was unable to demonstrate, after a period of 24-30 hours, the presence of virulent cholera organisms fed to fowls. On the other hand, it is not uncommon for cholera organisms, at least analogous organisms, to be isolated from fowls showing no evidence of cholera. In fact, these organisms have been advanced by some as the causative factors in diseases other than cholera. Weaver and Mitchell,[28] in their recent study of the etiology of roup in fowls, report the isolation of an organism of the pasteurella group which, when inoculated into fowls, produces a disease indistinguishable from roup. Some 8 to 10 years ago, Jackley,[29] working along lines similar to those of Weaver and Mitchell, reported the possibility of *P. avicida* as the causative factor in roup of fowls. Beach,[19] in his studies of fowl cholera in California, reports the presence of the organisms associated with ruptured oviduct in fowls.

Mode of Infection

The digestive tract is considered as the possible avenue in natural infection, though the experimental feeding of virulent

TABLE I—*Showing cultural and biochemical characteristics of 24 strains of P. avicida.* (Patton)

	Number of Strains	24
Morphology	Size in micra	0.5 x 1.2
	Motility	−
	Spores	−
	Gram stain	−
Cultural Features	Clouding of bouillon	23 + 1 Sl.
	Ring	7 + 17 −
	Pellicle	−
	Growth at room temp.	23 + 1 Sl.
	Growth at 37.5° C.	+
	Growth at 45° C.	22 + 1 − 1 Sl.
	Gelatin liquefaction.	23 − 1 Sl.
Biochemical Features	Milk: Coagulated	−
	Milk: Acidified	−
	Milk: Reduced	−
	Blood plates (hemolysis)	22 − 2 Sl.
	Fermentation: Dextrose AG	+ −
	Fermentation: Lactose AG	− −
	Fermentation: Maltose AG	− −
	Fermentation: Saccharose AG	− +
	Nitrates reduced	+
	Indol Ehrlich: First week	15 + 9 −
	Indol Ehrlich: Second week	24 +
	Pathogenicity for fowls	12 + 2 − 10 ○

− = Negative. + = Positive. Sl. = Slight. ○ = No test.

cultures to fowls rarely gives rise to the infection. Infection may take place when the resistance is greatly reduced or in the presence of various pathological conditions. It is entirely possible, however, that natural immunity occurs by way of the digestive tract. Infection may also occur through abrasions of the skin and mucous membranes. The work of Bordet and Fally[30] shows that it is possible also to infect, experimentally, by means of the conjunctiva, either by direct inoculations or the sewing of a string therein. Shilston[23] states that the organism may be conveyed by biting flies (hemorrhagic septicemia of cattle and wild game in India).

Pathogenesis

The bipolar organisms may penetrate the intestines and lymph spaces, thus gaining entrance to the blood-stream, where they multiply with great rapidity. When infection takes place through a breach in the continuity of the skin or mucous membrane, death results in a short time. Thiede[31] found bacilli in the internal organs of a mouse, 15 minutes after a subcutaneous injection. In connection with injections given intramuscularly, a degenerative area of necrosis (cooked meat appearance) at the point of the injection is of diagnostic importance and characteristic of the local pathological changes produced by this organism.

Cause of Death

Hutyra and Marek refer to the toxins formed in the animal body and the negative chemotaxis of the virulent organisms inactivating phagocytic action. This interpretation, as to how injury is produced, is based upon the work of Gabritschewsky, Silberberg, and Zeliony and their studies of negative and positive chemotactic action of virulent and attenuated bacilli, and on Pasteur's early work in the separation of the toxin and bacilli by filtration. This hypothesis is advanced by many of our texts and some of our writers, especially by those who have worked but little with this particular organism.

In a series of classic papers, which appear to have attracted less attention than they deserve among American students of immunological problems, Edward Weil,[32] following the steps of Bail, has presented the clearest evidence as to the elements favoring infection, which characterize the activity of the bacteria of the fowl cholera group. An analysis of the situation, as based upon his studies and those of his collaborators, Weil and Braun, may briefly be summarized as follows:

1. Rabbits inoculated into the pleural cavity with virulent fowl cholera cultures produce an exudate which harbors substances favoring infection—the aggressins.

2. These aggressins, which are manifestly equivalent to the virulins of Rosenow and the antiphagins of Tchistivitch and Yourevitch, possess in high degree the property of furthering infection.

3. The infection-furthering properties of the exudate do not lie in poisonous substances, since animals easily tolerate eight times the dose necessary to determine a fatal issue when administered together with a non-lethal dose of virus.

4. The results cannot be due to an inactivating influence in the opsonic sense, since leukocytes, whether in immune or normal animals, manifest no or only slight phagocytosis, either *in vitro* or *in vivo*.

5. Toxicity and aggressivity are not identical; toxicity is most noteworthy in organisms which are not highly aggressive; and the most aggressive organisms are non-toxic. *P. avicida* is one of the most aggressive organisms that have been studied.

Weil concludes that one can not demonstrate the slightest toxicity against the most susceptible animals, like the rabbit, in the case of fowl cholera. Citron and Putz,[33] who employed serum extracts and water extracts (artificial aggressins), furnish another line of evidence which demonstrates the lack, at least, of extracellular toxins.

There is further good evidence of the lack of toxin-production by this organism in the work of Hüntemuller.[34] One phase of this writer's experiments involved the injection into rabbits of large amounts of bacterial filtrates, in order to ascertain whether protective substances could be washed out of the cells. The point of interest here lies in the fact that the injection of large amounts of filtrates produced no ill effects. Hadley,[35] using his most virulent culture (48), the m.l.d. of which, for rabbits, was one-quintillionth of a cubic centimeter, had occasion to administer large doses of a killed, broth culture, heated at 63° C. for 30 minutes, a temperature considered too low to destroy *in toto* extracellular toxins, if present. This showed no toxicity for rabbits in the amount used. Hadley later demonstrated more conclusively the absence of extracellular toxins, as follows: A rabbit was inoculated with 0.001 cc of a 24-hour broth culture of strain 48, intra-abdominal route, and died in 14 hours. A culture was regained from the heart-blood and plated. A colony was subcultured into chicken broth, grown at 37° C. for 48 hours and passed through a Berkfield N candle under suction. The filtrate was tested for sterility by inoculating 10 tubes and growing for 96 hours. No growth appeared at the end of that time. The remaining portion was divided into three lots, one portion incubated for 24 hours, another placed in the ice-box, and the third heated at 62° C. for twenty minutes. These were

injected intravenously, in amounts of 1 cc, into rabbits, all of which remained normal. If I interpret the available literature correctly, together with my own observations, the possibility of the *P. avicida* being a toxin producer, in the sense that we commonly understand and apply the term, is remote. As to whether or not these organisms produce an extracellular toxin in the animal body, I would not attempt to hazard an opinion.

Resistance of the Organism

According to Gaertner,[36] the organisms may remain alive in manure for three months; in putrefactive carcasses and garden soil for three months (Kitt[37]); in water, with the exclusion of air and at a temperature of 5-6° C., for 18 days (Hertel[22]). To the influence of cold, they show considerable resistance, resisting freezing for 14 days (Kitt[37]), while Hertel[22] found the resistance considerably reduced at minus 13 degrees. Towards other harmful influences, they appear less resistant. Thus, when dried in the air and subject to sunlight, they lose their virulence in 48 hours, and in excluded light in 73 hours (Helfer[38]). When subjected to a temperature of 45-59° C. for 45 minutes or 80-85° C. for 10 minutes, the cultures are sterile. Salmon[39] found 0.5 per cent sulphuric acid and one per cent carbolic acid destructive to the organisms. According to Colin,[40] the blood and loose excrements are disinfected by thoroughly mixing them with a five per cent copper sulphate solution.

Virulence of the Organisms

Hadley and Amison,[6] working with their culture 48, determined the m.l.d. as follows for the various animals:

Table III—*Virulence of P. avicida as determined by Hadley*

Fowls	0.000,000,000,000,001 cc (one-quadrillionth of 1 cc)
Pigeons	0.000,000,000,000,000,001 cc (one-quintillionth of 1 cc)
Turkeys	A 23-pound turkey failed to survive a dose of 1 cc
Ducks and Guineas	1 cc proved fatal in 18 hours
Rabbits	0.000,000,000,000,000,001 cc (one-quintillionth of 1 cc)

Hadley's culture 48 appeared to be extremely virulent, much more virulent than is ordinarily obtained. In the writer's experience, one that would average 0.1-0.01 cc of a 48-hour culture would be considered virulent.

Variability of the Virulence

This group of organisms, of which the fowl cholera organism is one, is found widely distributed in nature, occurring as saprophytes in the soil, in slow-moving or stagnant water, on all kinds of dead organic matter, and on various plants. Under normal conditions, they do not manifest pathogenic characteristics. They occur not infrequently in the digestive and respiratory tracts of fowls. Under conditions not well understood, they assume virulence and pathogenicity and become capable of successfully invading the tissues of apparently healthy fowls. Their pathogenicity is favored by those factors which might be considered as predisposing: age, colds, exertion, anemia, parasites, mixed infections, and the like. Parasites in the respiratory and digestive tracts, by injury to the epithelium of the mucous membranes, may facilitate entrance and infection. Organisms which have become pathogenic may return to their non-pathogenic existence upon leaving the animal body. Most likely, however, after becoming accustomed to their host, they obtain an increased virulence for these animals, even in the absence of predisposing factors. These characteristics of the organisms explain, in part, the observed variability in virulence of the organisms. The common experience is that the disease appears periodically and spontaneously in many instances. In part, this may be explained by the sudden increase in virulence of the organisms present in the soil or elsewhere, favored by pre-

Fig. 1. Chickens with fowl cholera sit quietly, with the neck contracted and the eyes partly closed

disposing influences. In other cases, although the disease may appear spontaneously, the organisms which pass through the fowls' bodies retain their virulence for a considerable length of time, thus spreading the disease by direct or indirect infection, and disseminating the organisms to distant localities.

Susceptibility

Fowls: The blood of fowls affected, as well as fresh cultures, produce the disease following inoculation. The domestic hen, turkey, pigeons, ducks, geese, pheasants, and other fancy birds are easily infected, as are many of the free-flying birds. Death often occurs in 12-24 hours following inoculation. However, it is not unusual to find fowls surviving several days.

Rabbits: These animals are most susceptible and very easily infected. Death generally occurs in the early stages of the disease.

Mice and rats: These are also susceptible, as is the ground squirrel. It was Metchnikoff's suggestion that the bacillus of fowl cholera be used to start true epizootics amongst the latter, in order to diminish their numbers in parts of Southern Russia.

Guinea pigs: These are fairly immune, except when the organisms have been passed through these animals, after which they may be infected by intravenous inoculation.

Dogs, cats, and swine: Relatively immune, except when the organisms have been passed through these animals, and then, with difficulty, may they be infected.

Sheep: Also relatively immune, except to virulent cultures given intravenously.

Anatomical Changes

In fulminating cases, either in natural or artificial infection, a lesionless condition may exist. In less acute cases, the principal anatomical changes observed are those of hemorrhagic inflammation, petechiation of the heart, intestines, and congestion of the lungs. The blood-vessels in some of the loops of the intestines may be injected. The entire duodenal portion of the intestine, sometimes the entire small intestine, viewed as a whole, unopened, has a distinctly pinkish coloration. Upon opening the duodenal, mucous membrane exhibits a petechiated condition, and the intestinal contents are fluid and usually of a greenish-yellow color, and may or may not be bloody. The serous contents of the pericardium are usually cloudy, and often exhibit the presence of small fibrinous flakes. In the writer's opinion, this is of some

diagnostic importance. The upper air passages may show some inflammation. Glandular organs often exhibit parenchymatous degeneration. As would be expected, the number and severity of the anatomical changes are in proportion to the length of time that the fowl lives.

Symptoms

In fulminating cases, there are generally no symptoms observed. In fact, many of these cases are found dead in the nest, on, or under, the roosts, or elsewhere. If, however, the disease is of less severity and the fowl dies 18-24 hours after infection, the following symptoms may be noticed: ruffled plumage, loss of appetite, thirst, disinclination to move, cyanotic comb, difficult respiration, trembling, a comatose condition, and high

Fig. 2. The acute type of fowl cholera kills quickly. Chickens that seem healthy may be found dead within a few hours.

temperature. The fowl dies finally after a few feeble convulsions. Greenish diarrhea is usually noticed. In the more protracted cases, the above symptoms are less severe. The fowl may assume a staggering gait, arthritis usually being present. The lameness might be confused with tuberculosis or gout. Emaciation may be observed as the disease progresses, the fowl becomes comatose and dies after a few convulsions, or recovers after a few weeks. Temperature is quite a reliable index as to the severity of the infection. Many affected fowls carry a temperature of 110° F., or higher. When the temperature goes above 108.5° F., very few, if any, recover.

Diagnosis

Fowl cholera, in its course and symptoms, resembles fowl plague, from which it may be differentiated upon bacteriological

examination by the presence of bipolar staining organisms and the absence of a filtrable virus. Fowl typhoid may easily be mistaken for cholera. In the former, the characteristic pinkish coloration of the intestines and musculature is replaced by a paleness. The heart and liver are often studded, in the case of typhoid, with pin-point, grayish areas of necrosis, cellular infiltration, or mahogany-colored liver. Fermentation reactions assist, as do also animal inoculations, the rabbit being most susceptible to cholera. Cholera-like diseases may be differentiated by bacteriological examination.

Prognosis

The outcome of an outbreak depends to a large extent upon the virulence of the invading organisms and the presence of predisposing factors, together with the methods of sanitation, quarantine, and treatment, both immunological and medicinal being practiced. In a natural infection, the organisms may successfully infect but few birds in a flock. The finding of a bird here and there, apparently well one hour and dead the next, continuing for several days, or weeks, is suspicious evidence of cholera, though it might pass unnoticed and the losses be attributed to something else. On the other hand, it might assume all of the proportions commonly attributed to it. In the former case, the veterinarian most likely would not be consulted, unless it assumed greater virulence, which it is potentially capable of doing. It is this aspect of the disease which necessarily makes the prognosis guarded, even when but few individuals in the flock are infected. There is little doubt that many, possibly as high as fifty per cent of the adult flock, would show considerable immunity to any but a most virulent strain of the organism. In less mature birds, possibly including yearlings, the degree of resistance is much less marked. And so on, down to the growing stage, under four months, where the resistance, even to an organism of slight virulence, is practically nil.

Treatment

At the onset of the disease, the treatment of the affected, and possibly the exposed, with potent immune serum would appear to be the logical course to pursue. Bacterins may assist materially in some instances, while in others they appear to be contraindicated.

We need to recognize three conditions in relation to the struggle between the host and the invading organisms:

1. That in which the host possesses sufficient immunity to localize and destroy the invading organisms rapidly, in which case treatment is not necessary.

2. That in which the invading power of the organism and the reactive power of the host are more nearly equalized and the infection tends to become localized and perhaps chronic, in which case bacterins may swing the balance in favor of the host.

3. That in which there is the entire lack of ability, on the part of the host, to localize and destroy the invading organisms. Bacterins may, if used before the infection takes place, assist in increasing the resistance of the host, but are of no value in con-

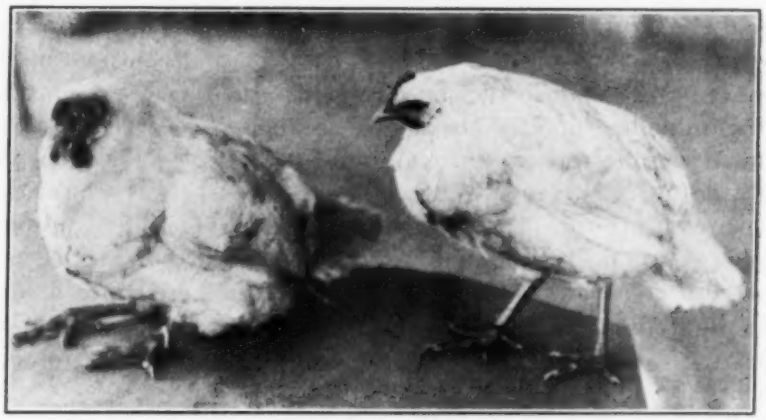

FIG. 3. Chickens with chronic type of fowl cholera. When affected with the chronic type of the disease, chickens lose their appetites and develop diarrhea. Their feathers are ruffled and their wings and tail feathers droop.

trolling the disease after infection takes place. As is well known, all bacterins (vaccines) produce active, acquired immunity, which, for success, depends upon the reactive power of the animal to form antibodies. If this always occurred following the inoculation of an antigen, vaccination would be much more of a success.

The reasons for the failure always to do so are obscure. This failure may not always be due to the bacterin, but may be, and often is, due to the lack of reactive power on the part of the host, or the inability of the individual to produce sufficient antibodies to overcome the invading organisms. Otherwise perfectly normal animals appear to lack this ability. On the

other hand, failure may be due to the bacterin, where the reasons for failure are numerous. Chief amongst these are:

1. Organisms may lack antigenic properties.
   A. Nucleo-proteins.
   B. Insolubility of proteins.
2. Mode of preparation destroys antigenic properties.
   A. Racemized protein.
   B. Protein rendered insoluble.
3. Protein hypersensitization.

As different strains of the same organism will vary considerably in their ability to produce antibodies, as outlined above, others may sensitize rather than immunize. To draw the line between sensitization, we would not have immunization, as it appears to be a step towards immunity. Under certain conditions, however, this stage of immunization may lead to unfavorable results. When the exfoliating stimulus is lacking, or has not occurred, and the antibody remains attached to the cell, this cell may be considered in a state of hypersensitization and continue in this state until the antibodies become free in the blood stream, when the individual may be considered immune. The introduction of more specific foreign protein, while the cell is still laden with sessile (attached) antibodies, will cause injury to the individual treated, since it is attached to the cell very rapidly and completely. A protein which stimulates the cell to produce only sessile (attached) antibodies can not be prepared into a satisfactory bacterin. Perfect results from vaccine treatment should not be expected. There will always be some failures from one cause or another.

Prevention

Care should be exercised in order that the introduction of the disease by the purchase of new birds, birds returning from shows, contests, and the like, may be prevented. A careful examination and quarantine for a period of at least ten days should be practiced. Bury deep or burn carcasses of dead fowls. The droppings and litter should be handled in like manner. Customary sanitary precautions should be exercised, not overlooking the feeding and drinking utensils. Quarantine against itinerant peddlers, free-flying birds and animals is a wise precaution.

Immunological Investigations

Immunization with attenuated cultures: This method of vaccination is of great scientific importance, as it was Pasteur's memorable work with this organism that established the founda-

tion upon which our present methods of immunity are based. Pasteur's method of attenuation was accomplished by culitvating the organisms, in the presence of atmospheric air, for a period of about two months, or until the virulence had disappeared. A second method of attenuation was accomplished by cultivating them in like manner for a shorter period of time. This constituted his second attenuated vaccine. Pasteur's methods in the hands of other workers have been only partially successful. Cagny[41] and Hess[42] secured but partial success in attempting to repeat Pasteur's work. Lignieres[43] produced a potent vaccine from cultures reinoculated several hundred times, from agar to agar, at two-day intervals, as a result of which the cultures obtained uniform virulence. Kitt[37] found that the blood of fowls immunized against the organisms had an immunizing

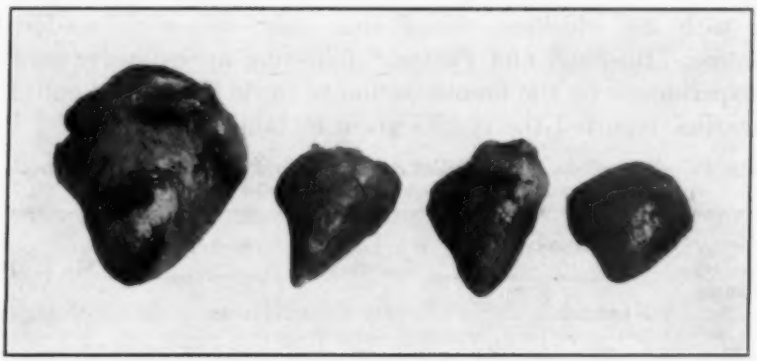

FIG. 4. Hemorrhagic lesions (petechiae) are characteristic of fowl cholera.

effect upon healthy fowls, and further concludes that the whites and yolks of eggs, from immunized fowls, had a similar protective action. Kitt and Mayer[44] were successful in preparing a serum, by the injection of virulent cultures into horses, which secured protection in rabbits, but which proved ineffective in fowls. These results were substantiated in part by Klett and Braun.[45]

Weil,[46] working with aggressins, after the method of Bail, was successful in producing an aggressin, a single injection of which protected guinea pigs and rabbits, as well as the fowl and pigeon, against a subsequent lethal inoculation. Citron and Putz,[33] working with serum and water extracts of cholera cultures, appear to have verified Weil's findings. Hadley and Amison[6] report one of their strains (52), almost avirulent, as producing

in rabbits the greatest immunity against their most virulent culture (48). Hadley[47] further reports producing complete immunity against his most virulent culture, with but a single inoculation of his almost avirulent culture (52). Hadley's results simulate those of Pasteur. Mack and Records[48] were unsuccessful in protecting fowls by the subcutaneous inoculations of bacterins, even when three treatments were given. Field tests appeared to offer some protection, in fifteen out of sixteen lots of fowl, in fourteen flocks. Unsuccessful results were also obtained in another flock, even following three treatments. Hadley and Caldwell[49] secured some protection in rabbits, with a 48-hour, heat-killed culture, against the inoculation of a virulent culture.

Van Es and Martin,[50] in their studies of seven commercial bacterins, with reference to their immunizing powers, and working with 200 chickens, found that only two were rendered immune. Bushnell and Patton,[51] following an extensive series of experiments on the immunization of fowls with killed-culture bacterins, reported the results given in table IV.

TABLE IV—*The effects of heat-killed cultures of a P. avicida in the control of experimental fowl cholera (Bushnell-Patton)*

No. of Birds	Survived		Controls	Survived		M. L. D.
	Number	%		Number	%	
58	36	61	20	4	20	4
42	23	55	47	0	0	5
33	14	42	10	0	0	25
68	25	37	7	0	0	50

Note.—One m. l. d. taken as that amount which killed all the test birds. Many birds were killed with a much smaller amount. Only those birds surviving for at least thirty days after receiving the virus were considered in these experiments.

The question of immunization appears to depend less upon the number and virulence of the organisms used in a bacterin, than upon the antigenicity of the organisms contained therein. Bushnell and Patton,[51] working on this phase, reported as shown in table V.

As to the advantages of fractional and single dosage in prophylactic inoculations, Bushnell and Patton,[51] working with *P. avicida* in experimental protection of fowls, reported the results shown in table VI.

TABLE V—*The variation in antigenic action of various strains of P. avicida (Bushnell-Patton)*

Vaccine No.	1	2	3	4	5	6	7	8	9	10	11	12	13	14	CONTROLS
No. Birds Survived	2	2	1	0	0	3	3	3	1	0	3	2	3	0	0

Note.—Three birds were tested against each bacterin. Vaccination consisted of inoculations of 2 cc of phenol-killed bacterin. Fourteen days later, 4 m. l. d. of virus was given. No cross vaccinations were attempted.

TABLE VI—*Advantages of single and fractional dosage in prophylactic inoculations. Fowls (Bushnell-Patton)*

BIRDS	TREATMENTS	VACCINE (CC)	M. L. D. OF VIRUS	SURVIVED	
				NUMBER	%
10	0	0	5	0	0
10	1	5	5	3	30
10	2	10	5	7	70
9	3	15	5	9	100

These results parallel those secured by Harvey and Iyengar,[52] working with *P. avicida* and experimental protection in pigeons.

TABLE VII—*Fractional and single dosage in experimental protection. Pigeons (Harvey-Iyengar)*

EXP.	NUMBER	TREATMENT	TESTED	RESULTS
1	58	None (Controls)	58	All died
2	15	One dose	12 days later	12 died
3	15	One dose	19 days later	11 died
4	15	Two doses	12 days later	7 died
5	15	Three doses	12 days later	1 died

Harvey and Iyengar conclude that two spaced doses of prophylactic antigen are better than a single dose, and three better than two. Development of protection following prophylactic inoculations. The work of Harvey and Iyengar[53] gives the following interesting data:

1. No evidence of protection is manifest seven days after a single dose of prophylactic antigen.

2. Development of immunity is significantly manifest very quickly after the administration of a second dose of prophylactic antigen.
3. Development of immunity reaches its maximum about the fourteenth day after the administration of a second dose of prophylactic antigen and remains at its maximum up to at least the thirtieth day.

With reference to the duration of the protection following prophylactic inoculations, some birds appear to develop a rapid immunity and retain it for a reasonably long period of time. In other cases, birds which have received treatment (bacterin) appear to become suceptible again in a short period of time. Bushnell and Patton[51] reported birds withstanding as much as 25 m.l.d. of a virulent culture ninety days following vaccination, whereas others, vaccinated at the same time and in like manner, failed to survive a much smaller amount.

Harvey and Iyengar[54] report the results of testing some eighty pigeons, which were vaccinated twice, with results shown in table VIII.

TABLE VIII—*Duration of protection following prophylactic inoculations (Harvey-Iyengar)*

DAYS AFTER LAST VACCINATION	SURVIVED*	
	TREATED	CONTROLS
30	6	0
90	6	1
150	1	0
180	1	0

*Ten pigeons treated and ten untreated as controls.

Harvey and Iyengar conclude:

1. That evidence of a high degree of protection is manifest for three months after inoculation of antigen.
2. That evidence of almost complete loss of protection is manifest five months after inoculation of antigen.

SEROLOGICAL TESTS IN RELATION TO IMMUNITY

Harvey and Iyengar[55] are of the opinion that, in all probability, there exists no correlation between agglutination content of the serum and immunity, in so far as fowl cholera is concerned. This is the opinion prevalent among those workers who have studied this phase of the problem, whereas the results of Bushnell and Patton[51] appear to show a reasonably close relationship between the complement-fixation test and immunity, as determined by the use of virulent cultures.

Acknowledgment

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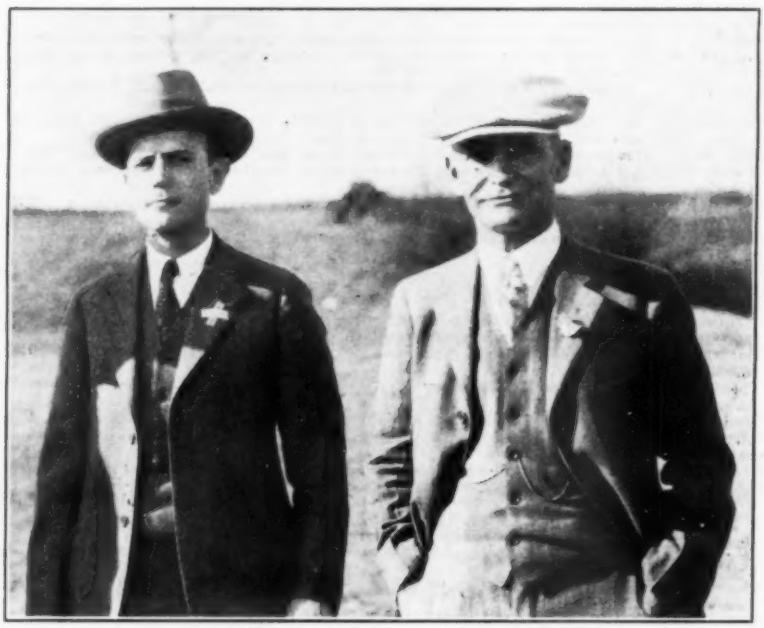
Between 55 and 60 million pounds of meat and meat products are consumed daily by the people of the United States, according to Oscar G. Mayer, president, Institute of American Meat Packers.



Officers of the Missouri Veterinary Medical Association: (left) Dr. Fred C. Cater, Sedalia, Secretary; (right) Dr. Hugh M McConnell, Independence, President.

STUDIES OF SALMONELLA PULLORA

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During the past few years, the diagnosis of *Salmonella pullora* infection, in baby chicks and adult fowls, has become an important problem. The following points were thought to be of importance as an aid in the diagnosis of this infection:

1. A study of the fermentative reactions of a number of strains of *S. pullora*.
2. A study of the interagglutinability of *S. pullora* with other intestinal organisms.
3. A study of the normal agglutinins of the blood of fowls.
4. The effect of phenol in the so-called pseudo-agglutination.

Since 1913, much has been added to our knowledge concerning *S. pullora*. Smith and Ten Broeck[1] carried out five sets of experiments with sera from rabbits that had been immunized with heated cultures of *Eberthella typhi* (*B. typhosus*), *Eberthella sanguinaria* (*B. sanguinarium*), and *S. pullora*. They decided that the agglutination tests were sufficiently definite to enable them to group *E. sanguinaria* and *S. pullora* together, both demonstrating the same intimate relation to *Eberthella typhi*.

Rettger and Koser[2] carried out agglutination tests, using reacting sera from rabbits immunized by subcutaneous injections. The first used killed suspensions and later living suspensions of *S. pullora* and *E. sanguinaria*. Five days after the injections of heated suspensions, the rabbits were bled, and the agglutinative powers of the sera were tested against definite suspensions of both *S. pullora* and *E. sanguinaria*. No difference in agglutination properties was apparent. Attempts were made to increase the agglutination titre by the injection of living organisms. The titre remained the same, and no change in the agglutinative ability of the two sera was manifest.

Taylor[3] concludes from his studies of fowl typhoid that the lesions produced in fowls which are infected with *E. sanguinaria* resemble, in many respects, those produced by *S. pullora*. There is a close resemblance in the biological character of the two organisms, but there is enough difference to warrant the conclusion that they are distinctly different diseases.

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Ward and Gallagher[4] studied forty-seven birds for the purpose of comparing agglutination and intradermal tests on naturally infected birds. They report the absolute failure of each test, as judged by the other test and an autopsy. Field tests on two hundred thirty-one birds, made simultaneously with the agglutination test, failed to detect any case reacting to the intradermal that was reported positive to the agglutination test.

Goldberg[5] demonstrated that the principal differences between the strains of *S. pullora* and *E. sanguinaria* lie in the fact that the former produces gas in various carbohydrates, while the latter lacks this power. The carbohydrates used consisted of the following: dextrose, lactose, saccharose, mannite, dextrine, inulin, galactose, levulose, raffinose, amygdalin, arabinose, adonite, dulcite, xylose, salicin, isodulcite, mannose, starch, glycerin and erythrol.

Hadley[6] concludes from his studies on the colon-typhoid intermediates that, in the carbohydrate media used, known cultures of *S. pullora*, *E. sanguinaria*, *Pasteurella avicida* (*B. avisepticus*), *Salmonella paratyphi* (*B. paratyphosus A*) and *Salmonella schotmulleri* (*B. paratyphosus B*) manifest definite fermentative differences which justify regarding them as distinct species. There is a closer fermentative relation between *E. sanguinaria*, *S. paratyphi* and *S. schotmulleri*, than between the latter two and *S. pullora*. This is due to the fact that *S. pullora* is maltose-dextrine-dulcite-negative, while the other three are positive in these media. Hadley also finds that all the maltose-dextrine-dulcite-negative strains of *S. pullora* isolated from chicks have been aerogenic, and all isolated from adult birds were anaerogenic. In another paper,[7] this author concludes that gas production by *S. pullora* may depend upon whether the cultures are grown in dextrose extract or infusion broth. Propagating cultures for many years on artificial media does not cause them to lose their gas-producing ability.

Hadley[8] suggests that *S. pullora* appears to stand as a borderline group in the colon-typhoid intermediates, between the actual paratyphoids and the actual paracolons. He further suggests that, in order to facilitate bringing about some degree of order in the group of colon-typhoid intermediates, aerogenic forms be referred to the paracolon group and anaerogenic strains to the paratyphoid group.

Rettger and Koser[2] present data which indicate that dextrine, maltose and dulcite are attacked by *E. sanguinaria* with the

production of acid. *S. pullora* produces gas in dextrose and mannite, while *E. sanguinaria* does not produce gas in any of the carbohydrates. These authors conclude that *S. pullora* manifests itself only as the cause of natural epizootic infection in young chicks, while *E. sanguinaria* attacks fowls of different ages, and is of no significance as the cause of disease in very young chicks.

Mulsow[9] concludes from his studies that *P. avicida* may generally be distinguished from *E. sanguinaria* by its action in milk, hydrogen sulphid production, fermentation of the carbohydrates, agglutination reaction and its pathogenesis. *S. pullora* and *E. sanguinaria* produce hydrogen sulphid in lead acetate media and a temporary acidity in milk. *P. avicida* does not produce hydrogen sulphid in lead acetate media nor is it acid in milk. It does not ferment any of the carbohydrates. According to this author, *S. pullora* may be distinguished from *E. sanguinaria* by the inability of the former to ferment dulcite, while the latter does ferment it. *E. sanguinaria* produces acid in maltose and does not produce gas in any of the carbohydrates. Rhamnose is fermented by *S. pullora*, while *E. sanguinaria* produces acid only after forty-eight hours of incubation.

Krumwiede and Kahn [10] report results which indicate that the essential characteristic of the paratyphoid-enteritidis group is the ability of its members to produce acid from rhamnose, differentiating both the aerogenic and anaerogenic members from *E. typhi*.

Gage[11] concludes from his data that *S. pullora* is maltose-dextrose-dulcite-negative and aerogenic, while *E. sanguinaria* is maltose-dextrine-dulcite-positive and anaerogenic. These characteristics are constant, and in making a diagnosis of *S. pullora* infection all cultures should be submitted to the maltose-dextrine-dulcite test and checked by gas production in dextrose. He decided that Hadley is correct in his contention that *S. pullora* is aerogenic when isolated from young chicks, and is anaerogenic when obtained from adult hens. Cultures from eggs have always been aerogenic. He demonstrates that there is an interagglutinability of *S. pullora* with *E. sanguinaria*, *E. typhi*, *S. paratyphi*, and *S. schotmulleri*. This has a tendency to make the test lose in specificity. He concluded, however, that the agglutination test is at present the best method for locating *S. pullora* infection and furnishing the poultryman a starting-point for its elimination.

The Fermentative Reaction of a Number of Strains of Salmonella Pullora

The cultures of Salmonella pullora were grown in test-tubes of uniform length and caliber and in infusion broth containing one per cent of the carbohydrate (Pfanstiehl.) Durham double-barreled fermentation tubes were employed and the cultures were observed daily for a period of seven days. Cultures were kept at 37.5° C. Twelve strains of the organism were obtained from various parts of the United States. Two drops of a bouillon suspension of each strain were used as the inoculum. In all, twenty-one carbohydrates were used, namely: glycogen, xylose, glycerin, maltose, lactose, sucrose, salicin, rhammose, mannose, galactose, dextrose, arabinose, adonite, and erythrol.

From the data in table I, it is apparent that S. *pullora* is positive in dextrose, xylose, levulose, mannite, rhamnose, mannose, galactose, arabinose. It is negative in glycerin, glycogen, maltose, adonite, dulcite, lactose, dextrine, sucrose, inulin, erythrol, raffinose, sorbite and salicin. In the latter there is a slight indication of fermentation in some strains of this organism. In xylose, all strains showed a slight acidity but none produced marked quantities of acid. All strains produced gas in dextrose and mannite, which is a characteristic property of this organism, this being a great aid in differentiating it from *E. sanguinaria*, as the latter does not produce gas in any of the carbohydrates.

Table II is a comparison of the fermentative activities of *S. pullora* with other organisms of the intestinal group. It will be noted in this chart that *S. pullora* does not ferment lactose or maltose. This enables a differentiation from *Escherichia coli, E. typhi, S. enteritidis, S. paratyphi, S. schotmulleri, and E. sanguinaria*, as these organisms ferment maltose quite readily and, in addition, *E. coli* produces both acid and gas in lactose.

It would seem, therefore, that in making a differential diagnosis of *S. pullora*, it is essential to ascertain its fermentation activities in dextrose, mannite, lactose and maltose.

A Study of Interagglutinability of Salmonella Pullora with the Colon-Typhoid Intermediates

The agglutination test has become a popular means of recognition of those individuals which have contracted *S. pullora* infection. These fowls may have become, through the presence of infection in their ovaries, carriers of infection to the offspring. This test is at the present time the best known means of eradi-

cating bacillary white diarrhea, which affects baby chicks. There are certain factors which influence this test and which suggest the need of modification of the technic to secure a higher degree of specificity. Three factors which seemed to be of prime importance, from this viewpoint, were: interagglutinability, normal agglutinins, and the so-called pseudo-agglutinations.

During the past few years there has been a question as to the minimum dilution which would be safe to apply in the agglutination test for *S. pullora* infection. Dilutions of from 1-10 to 1-100 have been used as the minimum and this variation has not been conducive to the standardization of this test. It was with the hope that a safe minimum could be established that the following was done.

Six strains of *S. pullora* were used in this work and one strain each of *E. coli*, *S. enteritidis*, *E. typhi*, *S. paratyphi*, *S. schotmulleri*, *S. suipestifer and E. sanguinaria.*

The antisera were produced by injecting rabbits with suspensions of the organisms in physiological sodium chlorid solution, which contained one-fourth to one-half per cent of phenol. The antigens were prepared by growing the organisms on veal-infusion agar for forty-eight hours, at a temperature of 37.5° C. The growth was washed with 15-20 cc of sterile saline solution and phenol was added in sufficient quantity to give the desired dilution. The suspension was filtered through a glass-wool bacterial filter and placed in sterile containers until used.

Two rabbits were used with each culture. Before making any injections, a small quantity of blood was drawn from each rabbit and the serum tested for antibodies against each antigen. No normal antibodies were observed. The injections were made intravenously into the marginal vein of the ear. When the serum titre was 1-500, or higher, the rabbits were bled and the sera saved.

A study of the tables outlining the results of the interagglutinability of *S. pullora* with other intestinal organisms reveals several interesting facts. It will be noted from table III, that cultures of *S. pullora* were agglutinated by all the sera used, excepting that of *E. coli* and *S. suipestifer*, which gave negative results in all instances. *E. typhi* serum completely agglutinated all six strains of *S. pullora* at a titre of 1-40. Several strains were partially agglutinated at 1-60 and one strain was agglutinated at 1-80. *S. enteritidis* serum gave complete agglutination of the six strains at 1-10 and with cultures E and F gave a partial

TABLE I—*Summary of data as regards fermentation of twelve strains of Salmonella pullora*

CULTURES	DEXTROSE		GLYCOGEN		XYLOSE		GLYCERIN		MALTOSE		LACTOSE		SUCROSE		SALICIN		SORBITE		DULCITE		INULIN		LEVULOSE		DEXTRIN		RAFFINOSE		MANNITE		RHAMNOSE		MANNOSE		GALACTOSE		ARABINOSE		ADONITE		ERYTHROL	
	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS
A	+	+	–	–	+	–	–	–	–	–	–	–	–	–	+	+	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	–	–	–	–	–	–
B	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	–	–	–	–	–	–
C	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
D	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
E	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
F	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
G	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
H	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
I	+	+	–	–	+	–	–	–	–	–	–	–	–	–	+	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
J	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
K	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–
L	+	+	–	–	+	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	–	+	–	–	–	–	+	+	+	+	+	+	+	–	–	–	–	–

TABLE II—*A comparison of the fermentation activities of Salmonella pullora with other intestinal organisms*

CULTURES	DEXTROSE		GLYCOGEN		XYLOSE		GLYCERIN		MALTOSE		LACTOSE		SUCROSE		SALICIN		SORBITE		DULCITE		INULIN		LEVULOSE		DEXTRIN		RAFFINOSE		MANNITE		RHAMNOSE		MANNOSE		GALACTOSE		ARABINOSE		ADONITE		ERYTHROL	
	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS	ACID	GAS
E. coli	+	+	-	-	-	-	-	-	+	+	+	+	-	-	-	-	+	+	+	+	-	-	+	+	+	+	-	+	+	+	+	+	+	+	-	-	-	-	-	-
E. typhi	+	-	-	-	-	-	-	-	+	-	-	-	-	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	-	-	+	-	+	-	-	-	-	-	-	-
S. enteritidis	+	+	-	-	+	+	-	-	+	+	-	-	-	-	-	-	+	+	+	+	-	-	+	+	+	+	+	+	-	-	+	+	+	+	-	-	-	-	-	-
S. paratyphi	+	+	-	-	-	-	-	-	+	+	-	-	-	-	-	-	+	+	+	+	-	-	+	+	+	+	+	+	-	-	+	+	+	+	-	-	-	-	-	-
S. schotmulleri	+	+	-	-	+	+	-	-	+	+	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	+	+	-	-	+	+	+	+	-	-	-	-	-	-
E. sanguinaria	+	-	-	-	+	-	-	-	+	-	-	-	-	-	-	-	+	-	+	-	-	-	+	-	+	-	+	-	-	-	+	-	+	-	-	-	-	-	-	-
S. suipestifer	+	+	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	-	+	+	+	+	-	-	+	+	+	+	-	-	-	-	-	-
S. pullora	+	+	-	-	+	-	-	-	+	+	-	-	-	-	-	-	-	-	+	+	-	-	+	+	+	+	+	+	-	-	+	+	+	+	-	-	-	-	-	-

TABLE III—*Agglutination of Salmonella pullora*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	–	–	–	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+	–	–	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–
S. paratyphi	+ + +	+ + +	+ + +	+ + +	+ +	+	–	–	–	–	–	–
S. schotmulleri	+ + +	+ + +	+ + +	+ + +	+ +	+	–	–	–	–	–	–
S. suipestifer	–	–	–	–	–	–	–	–	–	–	–	–
E. sanguinaria	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora A	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora B	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora C	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora D	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora E.	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora F	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE IV—*Agglutination of Eberthella sanguinaria*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	–	–	–	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+ +	–	–	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–	–
S. paratyphi	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–	–
S. schotmulleri	+ + +	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–
S. suipestifer	–	–	–	–	–	–	–	–	–	–	–	–
E. sanguinaria	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. pullora A	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–
S. pullora B	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–
S. pullora C	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–
S. pullora D	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–
S. pullora E	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+
S. pullora F	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE V—*Agglutination of Salmonella enteritidis*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	–	–	–	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
E. typhi	–	–	–	–	–	–	–	–	–	–	–	–
S. paratyphi	–	–	–	–	–	–	–	–	–	–	–	–
S. schotmulleri	–	–	–	–	–	–	–	–	–	–	–	–
S. suipestifer	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–
E. sanguinaria	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–
S. pullora A	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. pullora B	+ + +	+ +	–	–	–	–	–	–	–	–	–	–
S. pullora C	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–	–
S. pullora D	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. pullora E	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. pullora F	+ + +	+ +	–	–	–	–	–	–	–	–	–	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE VI—*Agglutination of Eberthella typhi*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	+ + +	+ + +	–	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. paratyphi	+ + +	+ + +	+ + +	+ +	+	–	–	–	–	–	–	–
S. schotmulleri	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–
S. suipestifer	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–	–
E. sanguinaria	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–
S. pullora A	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–
S. pullora B	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	+	–	–	–	–
S. pullora C	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–
S. pullora D	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–
S. pullora E	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–
S. pullora F	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE VII—*Agglutination of Salmonella paratyphi*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	+ + +	+ + +	–	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+ + +	+ + +	+ + +	–	–	–	–	–	–	–
S. paratyphi	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. schotmulleri	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–
S. suipestifer	+ + +	+ +	–	–	–	–	–	–	–	–	–	–
E. sanguinaria	+ + +	+ + +	+ + +	+	–	–	–	–	–	–	–	–
S. pullora A	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–	–
S. pullora B	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–	–	–	–
S. pullora C	+ + +	+ + +	+ + +	+ + +	+ + +	–	–	–	–	–	–	–
S. pullora D	+ + +	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–
S. pullora E	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–	–	–	–
S. pullora F	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE VIII.—*Agglutination of Salmonella schotmulleri.*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+ + +	+ + +	+ + +	–	–	–	–	–	–	–
S. paratyphi	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	–	–	–	–	–
S. schotmulleri	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. suipestifer	+ + +	+	–	–	–	–	–	–	–	–	–	–
E. sanguinaria	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–
S. pullora A	+ + +	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–	–	–
S. pullora B	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–
S. pullora C	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–	–
S. pullora D	+ + +	+ + +	+ + +	+ + +	+ + +	+	–	–	–	–	–	–
S. pullora E	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–	–
S. pullora F	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ +	–	–	–	–	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE IX—*Agglutination of Escherichia coli*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
S. enteritidis	–	–	–	–	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. paratyphi	+ + +	+	–	–	–	–	–	–	–	–	–	–
S. schotmulleri	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. suipestifer	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–	–
E. sanguinaria	+ + +	+ +	–	–	–	–	–	–	–	–	–	–
S. pullora A	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora B	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora C	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora D	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora E	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora F	–	–	–	–	–	–	–	–	–	–	–	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

TABLE X—*Agglutination of Salmonella suipestifer*

POSITIVE SERA	DILUTIONS											
	1–5	1–10	1–20	1–30	1–40	1–50	1–60	1–80	1–100	1–150	1–200	1–250
E. coli	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–	–
S. enteritidis	+ + +	+ + +	–	–	–	–	–	–	–	–	–	–
E. typhi	+ + +	+ + +	+ + +	–	–	–	–	–	–	–	–	–
S. paratyphi	+ + +	+ + +	+	–	–	–	–	–	–	–	–	–
S. schotmulleri	+ + +	+ + +	+ +	–	–	–	–	–	–	–	–	–
S. suipestifer	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +	+ + +
E. sanguinaria	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora A	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora B	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora C	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora D	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora E	–	–	–	–	–	–	–	–	–	–	–	–
S. pullora F	–	–	–	–	–	–	–	–	–	–	–	–
Normal serum	–	–	–	–	–	–	–	–	–	–	–	–

Complete agglutination + + +
Two-thirds agglutination + +
One-third agglutination +
Negative agglutination –

agglutination at 1-20. *S. paratyphi* and *S. schotmulleri* sera agglutinated the six cultures at practically the same dilution, 1-40.

The interagglutinability of *E. sanguinaria* and *S. pullora*, as shown by table IV and the one just studied, is very interesting. *E. sanguinaria* serum agglutinates *S. pullora* antigen quite as well as it agglutinates its homologous antigen and *S. pullora* serum agglutinates *E. sanguinaria* antigen in the same manner. Table IV shows that *S. enteritidis*, *E. typhi*, *S. paratyphi*, and *S. schotmulleri* sera all agglutinate *E. sanguinaria* antigen at about the same titre as they agglutinate *S. pullora*. *E. coli* and *S. suipestifer* failed to agglutinate *E. sanguinaria* antigen.

The agglutination of *S. enteritidis* is shown in table V. This antigen was agglutinated at a titre of 1-10 by sera of all six strains of *S. pullora*. It was agglutinated at 1-80 by serum from *E. sanguinaria* and at 1-200 by serum from *S. suipestifer*. It was not agglutinated by serum from *E. coli*, *E. typhi*, *S. paratyphi* or *S. schotmulleri*.

E. typhi, as shown in table VI, is agglutinated by serum from all the organisms used, the agglutinative titre of *S. pullora* being the highest (1-80).

The results of the agglutination of *S. paratyphi* and *S. schotmulleri* are given in tables VII and VIII. It will be noted from these that the two organisms were affected in practically the same degree by the different sera. All of the sera agglutinated these antigens as follows: *E. coli*, 1-10; *S. enteritidis*, 1-30; *E. typhi*, 1-40; *S. suipestifer*, 1-10; *E. sanguinaria*, 1-30; and the different strains of *S. pullora*, 1-40. The agglutinative titre of *S. pullora* serum for those two organisms is not as high as for *E. typhi*.

It will be noted in table IX that *E. coli* is not agglutinated very highly by the serum of any of the other organisms used. It is agglutinated by *E. typhi* and *E. sanguinaria* sera at a titre of 1-10. *S. paratyphi*, *S. schotmulleri* and *S. suipestifer* sera agglutinate this antigen at 1-20. *S. enteritidis* and *S. pullora* sera failed to agglutinate this antigen with the exception of culture B, which showed a partial agglutination at 1-10.

S. suipestifer, as shown in table X, is not agglutinated by the serum of *S. pullora*, or of *E. sanguinaria*. It is agglutinated by the sera of the other organisms used, at a titre of 1-20.

From the results obtained in the foregoing, it is apparent that there exists an interagglutinability between *S. pullora* and other intestinal organisms. This may reach a titre of 1-80 with the

sera of some of the organisms. It is evident, therefore, that it is not safe, in applying the agglutination test for *S. pullora* infection, to use a titre lower than 1-100.

The results obtained in the interagglutinability of *E. sanguinaria* and *S. pullora* indicate that the agglutination test for the latter is not specific, as serum from *E. sanguinaria* has a high agglutinative titre for *S. pullora* antigen. This, however, does not necessarily lessen the value of the practical application of this test, as it is quite as desirable to eliminate carriers of *E. sanguinaria* infection as those of *S. pullora*.

A Study of the Normal Agglutinins of the Blood of Chickens

In connection with the work done on the interagglutinability of *S. pullora* with other intestinal organisms, it was decided to ascertain whether the blood of chickens normally contained agglutinins for *S. pullora*.

The method used was as follows:

In all, eight birds were used. They were bled and the sera were tested with *S. pullora* antigen at titres of 1-5, 1-10, 1-20, 1-40, 1-60, 1-80 and 1-100. Five of the eight birds agglutinated in all the dilutions, while the remaining three gave positive results only in titres of 1-5 and 1-10.

The birds were killed and an examination of the ovaries made. The ovaries of the five birds which gave positive agglutination in all the titres contained some irregular and discolored ova which, on culture, revealed *S. pullora*. The ovary of one of the birds, which reacted only at 1-10, also contained some angular discolored ova and *S. pullora* was isolated from it. The ovaries of the two remaining birds, which gave positive agglutination only at 1-5 and 1-10, were normal in appearance and on culture were negative for *S. pullora*.

The number of birds used was not sufficient to arrive at definite conclusions, but the results obtained indicate that there normally exist, in the blood of chickens, agglutinins for *S. pullora*. From this viewpoint, a safe titre to use in the agglutination for bacillary white diarrhea should be well above 1-10.

The Effect of Phenol in the So-Called Pseudo-Agglutination

During recent years, thousands of fowls have been tested for *S. pullora* infection by means of the macroscopic agglutination test. In many cases, the fat content of the blood serum was so

heavy that the accuracy of the test was greatly impaired. Since the efficiency in the control of this infection depends upon the accuracy of the test, it is desirable that this factor be eliminated or controlled.

In experiments conducted with laying and non-laying hens, Warner and Edmond[12] find the fat content to be higher in the laying than in the non-laying fowls, both being fed the same ration. They state that fasting hens for sixteen hours brings about a decrease in the fat content of the blood.

Hitchner[13] conducted a series of experiments in which he found that, by fasting the birds for at least thirty-six hours previous to bleeding, the sera would be clear and result in excellent tests. He also, however, found that starvation caused a marked decrease in egg production which lasted about four days. This, in itself, makes it desirable that some other means be found to eliminate this pseudo-agglutination. There is serious objection to reducing the egg production in a flock, if it can be controlled in some other manner.

A series of agglutination tests was made in the usual manner, using the following mixtures: serum plus antigen and phenol 0.5 per cent; serum plus antigen with no phenol added; serum plus normal salt solution; serum plus normal salt solution and phenol 0.5 per cent. The sera from one hundred birds were used and each blood sample was mixed with the four solutions above mentioned.

When the tests were observed, after incubation, it was found that sixty per cent of those which contained serum, antigen plus phenol showed the so-called pseudo-agglutination. The tubes which contained serum, normal salt solution plus phenol showed results identical with those with the serum, antigen plus phenol. In the tests containing only serum and normal salt solution no change was apparent. The tests containing serum and antigen, with no phenol, revealed no pseudo-agglutinations.

The above routine was repeated a number of times and the same results obtained. For the past two years, in our laboratory, the agglutination test for *S. pullora* infection has been conducted without the use of phenol in our antigens and the pseudo-agglutination problem has been practically eliminated.

Summary

From the foregoing data the following conclusions appear justified concerning the diagnosis of *Salmonella pullora* infection in the fowl:—

1. In the identification of *S. pullora* it is essential, in addition to studying its special morphological characteristics, to ascertain its fermentation activities in dextrose, mannite, lactose and maltose.

2. There exists an interagglutinability between *S. pullora* and other intestinal organisms. This may reach a titre of 1-80 with the sera of some of the organisms. It is evident, therefore, that it is not safe, in applying the agglutination test for *S. pullora* infection, to use a titre lower than 1-100.

3. The results obtained in the interagglutinability of *E. sanguinaria* and *S. pullora* indicate that the agglutination test for the latter is not specific, as serum from *E. sanguinaria* has a high agglutinative titre for *S. pullora* antigen. This, however, does not necessarily lessen the value of the practical application of this test, as it is quite as desirable to eliminate carriers of *E. sanguinaria* infection as those of *S. pullora*.

4. It is evident that the blood of chickens normally contains agglutinins for *S. pullora*. The normal agglutinative titre is 1-10 and in the application of the agglutination test for this infection it would not be advisable to use a titre that was not well above this range.

5. The so-called pseudo-agglutinations in the application of the macroscopic agglutination test for *S. pullora* infection can be controlled by the use of antigens which have no phenol added as a preservative.

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NEURITIS IN CHICKENS

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The disease to be discussed here has been prevalent during the past few years. The term neuritis is not entirely free from objections, because in affected flocks there is such frequent association of myelitis, encephalitis, and iritis, that one is led

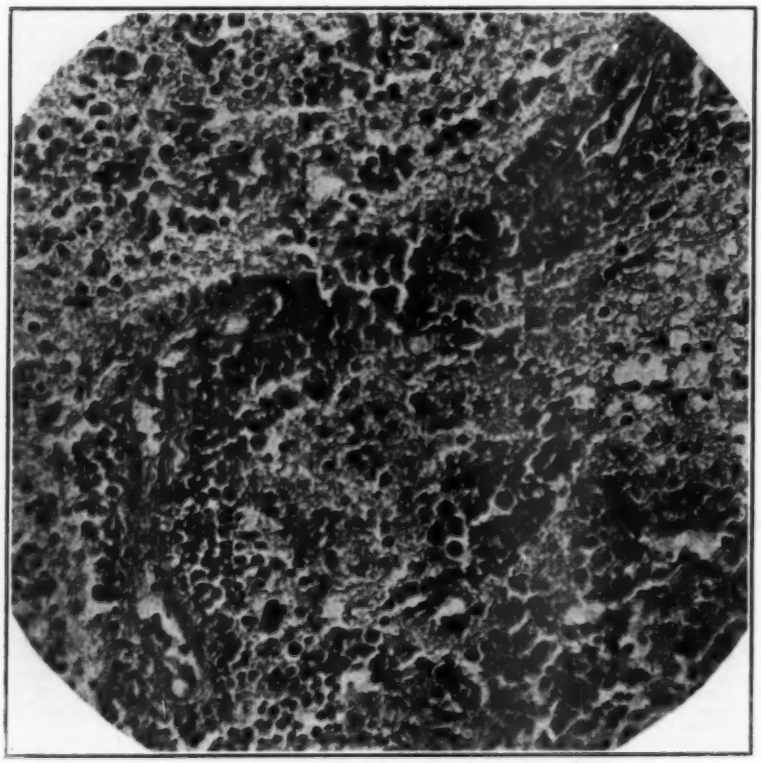

FIG. 1. Photomicrograph of a section of cerebrum, showing marked encephalitis. Note relation of infiltrating cells to blood-vessels.

to believe that the underlying disease is not limited to neuritic manifestations. Some of the symptoms of this disease are so similar to those produced by certain food deficiencies that nutritional factors have been suspected as the cause. This

[1]Received for publication, October 16, 1925.

suspicion has received confirmation from the fact that affected birds not infrequently recover when placed under favorable conditions and supplied with plenty of suitable food. As will be seen later, the pathology of the disease under discussion here is quite different from what is produced by a deficiency of antineuritic vitamin. This disease is of some importance in nutritional investigations with chickens, because its incidental occurrence may lead to erroneous conclusions regarding the antineuritic properties of the rations.

Aside from a few publications in which this disease is mentioned as being due to intestinal parasites, discussions of it seem to be rare in the literature of poultry disease. What appears to be the same disease was studied by Van der Walle and Winkler-

FIG. 2. The gross appearance of the sciatic nerve from a neuritic chicken. A part of the nerve is seen to be markedly swollen.

Junius,[1] in Holland. The gross and microscopic pathology of the spinal cord and peripheral nerves, as described by these two authors, corresponds with what has been found here. The results of inoculation experiments led the Holland investigators to believe that the disease is due to a filtrable virus. They considered "neuromyelitis gallinarum" to be a suitable name for the disease they studied. In 1907, Marek[2] reported finding inflammation of the nerves, more particularly the plexuses, of chickens showing paralytic symptoms. Upon examining the swollen nerves microscopically, he found a mononuclear cell infiltration.

The seriousness of the disease under farm conditions varies from a few cases in a flock to the actual loss of one-half or more

of the younger birds. It occurs mainly in birds from about four to eight months of age. Consequently, it is most prevalent during the summer and fall. However, cases do occur in birds as young as one month, and in some as old as one year or more. The symptoms shown depend upon the portion of the nervous system involved. If the sciatic nerve or some of its branches are involved, there will be a paralysis or at least an impairment

FIG. 3. A mature cock bird, showing the peculiar iritis ("white eye"), frequently associated with neuritis.

of movement of the corresponding muscles. It is common for affected birds to lose control of the legs, particularly from the hocks down, and then move about by flapping the wings. A striking feature of this form of the disease is the maintenance of a good appetite. The affected birds eat as long as they are able to reach the feed. In some cases, there is an apparently complete recovery, while in others, there is a gradual extension of the paralysis, resulting in complete helplessness, and finally death, in a state of extreme emaciation.

If the nerves of the wing are affected, there is a corresponding dropping of the wing. Involvement of the spinal cervical nerves may be associated with "wry-neck." Symptoms of myelitis alone occasionally occur, in which it appears that the damage is limited solely to the spinal cord. Inflammation of the vagi and other splanchnic nerves appears sometimes to produce inappetence and some other general symptoms, without the occurrence of paralysis. The encephalitis, which occasionally occurs, usually produces lethargic symptoms. The iritis, which at least has a flock association with this disease, is manifested finally by a contracted, non-responsive pupil and a strikingly whitish iris. The iritis develops slowly. In the final stages, the pupil becomes small, and the iris becomes whitish and fixed, so that the pupil does not respond at all to changes in intensity of light. In many

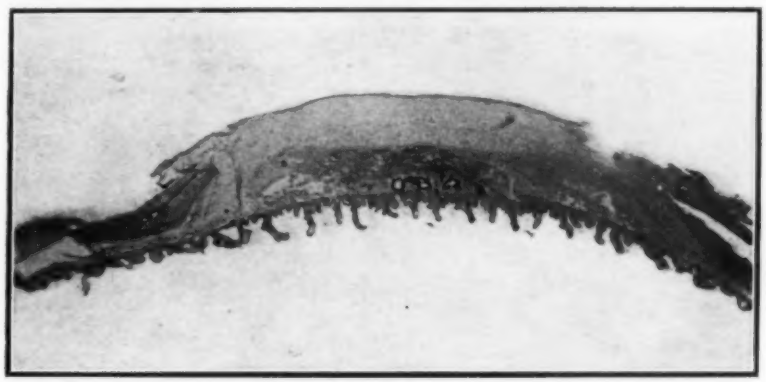

FIG. 4. Photomicrograph of a section of iris and cornea from a case of iritis. The iris is markedly infiltrated with mononuclear cells and is adherent to the cornea.

cases, the impairment of vision appears to be due solely to the contracted pupil and fixed iris.

The gross pathology is usually characteristic in at least some of its features. Swelling and grayish coloration of the large peripheral nerves is perhaps the most common and easily recognized lesion. If the bird shows paralysis of one or both legs, the corresponding sciatic nerve will usually show gross lesions, consisting of swelling and grayish coloration. The femoral nerves, the intercostal nerves, the cervical spinal nerves—in fact, any nerve trunk which is large enough to be examined grossly—may show well-defined lesions. An interesting feature of the pathology is the swellings which are frequently found in the vagi and other splanchnic nerves. The swollen portion of a nerve

may be extensive enough to involve practically the entire trunk, or it may be confined to a short segment. In some cases, the swelling and discoloration of the nerves are more conspicuous in the plexuses than anywhere else.

It is well, of course, to keep in mind thatt he important pathologic changes in individual birds may be limited to the central nervous system. A myelitis is often accompanied by enlargement of at least some of the spinal ganglia. In the absence of enlargement of the ganglia, it is difficult to recognize a myelitis on gross examination alone. An encephalitis can

FIG. 5. A young chicken in which the neuritis was apparently limited strictly to the nerves of the lower part of the legs and the toes. Microscopic sections from the nerves of this chicken showed the same kind of infiltration as is found in neuritis of older birds.

frequently be recognized by the presence of grayish-yellow foci in the brain, particularly in the cerebrum, and by the increased pressure on the cerebral fluid.

In the cases of iritis, a gross examination often shows adhesions between the iris and lens or the cornea. As a rule, postmortem examination of the eye does not show any other gross change in addition to what can be seen in the living bird.

Microscopic examination of affected nervous tissue shows a marked cellular infiltration. The infiltrating cell is fairly large, and is mononuclear. In peripheral nerves supplying skeletal

muscles, and in splanchnic nerves, the entire cross-section of the nerve trunk is often densely packed with the infiltrating cells to the practical exclusion of nerve fibers. In some nerves, however, the infiltrating cells have a patchy arrangement, which is apparently determined by the location of blood-vessels.

In the central nervous system, the infiltrating cells usually show an unmistakable perivascular arrangement. In sections of the spinal cord, the meninges often contain many mono-

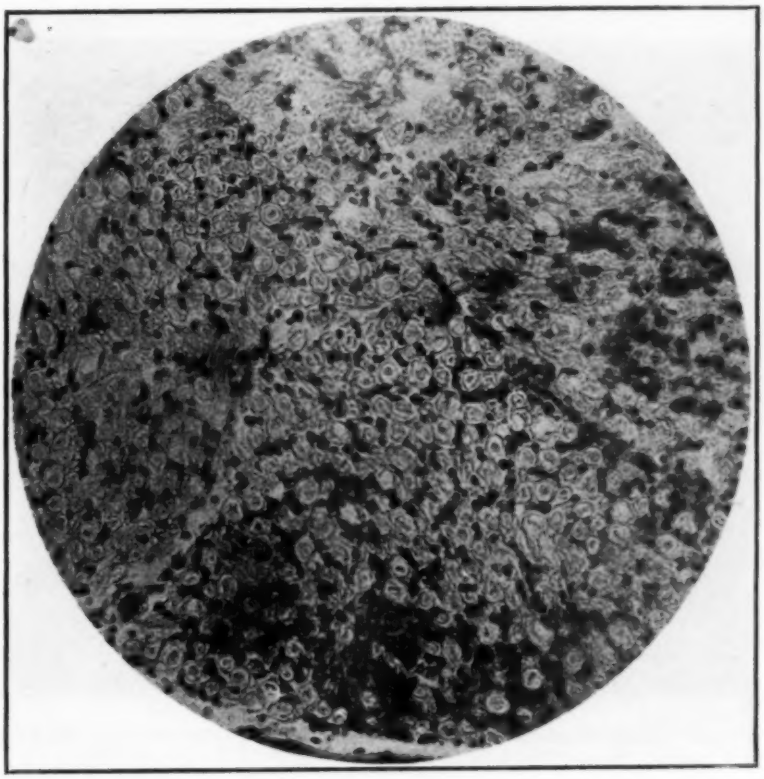

FIG. 6. Photomicrograph of a part of a cross section of a sciatic nerve, showing a patchy distribution of the infiltrating cells.

nuclear infiltrating cells which have predominantly a perivascular arrangement. In the substance of the cord, a mantle of mononuclear cells is frequently found around the blood-vessels. The roots of the spinal nerves commonly show the same type of cellular infiltration. Sections of brain from an encephalitic case show perivascular infiltration. Occasionally there is a diffuse distribution of the infiltrating cells accompanied by well-marked degenerative changes in the brain tissue.

Sections of an eye in the advanced stages of iritis show the iris to be packed with mononuclear cells and consequent marked dissociation of the muscle fibers.

The pathology of this disease, of course, strongly suggests that it is infectious in nature. In our experiments, however, we have not succeeded in reproducing it by parenteral injections or by feeding tissues from affected birds. In one series of experiments, seventeen chickens were injected subcutaneously, and eleven were injected intracranially, with material from eleven

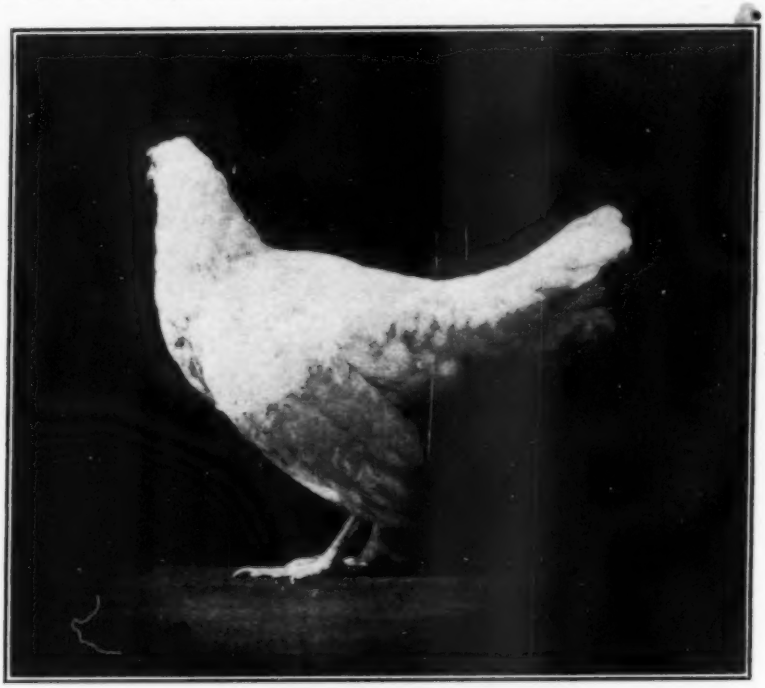

FIG. 7. A drooping wing, due to inflammation of the nerves. The bird also shows general symptoms suggesting a more extensive involvement of the nervous system.

different field outbreaks. The material injected consisted of physiological salt solution washings from triturated sciatic nerves, vagi nerves, spinal cords, and brains from birds showing typical symptoms and lesions. Only nerves showing unmistakable lesions were selected for trituration. The birds injected ranged in age from two to six months; the majority were four to five months of age.

One of the birds injected subcutaneously died three days after injection. Autopsy showed old, liver lesions. Four of the other birds injected subcutaneously, and three of those injected

intracranially, died within from one to five months after injection. The other twenty birds all remained well for at least five months, some of them for at least eight months. None of the birds that died following injection showed any symptoms or gross lesions suggestive of the disease under investigation. Microscopic examination was made of the brain, cord, and peripheral nerves from four of them. No microscopic lesions were found.

In another experiment, four five-months-old pullets were injected as follows: One was injected intraperitoneally and subcutaneously; and another was injected intracranially with brain, cord, and markedly enlarged sciatic nerves from a typical case of the disease which had been affected for ten days. Another

FIG. 8. A characteristic position of a bird in an advanced stage of the disease. The pronounced paralysis and the lethargic symptoms suggest an extensive involvement of the nervous system.

pullet was injected subcutaneously and intraperitoneally; and still another was injected intracranially with sciatic and vagi nerves from two field cases showing excellent gross lesions in the sciatic and vagi nerves. The four pullets were then kept under observation for five months, during which time they did not show symptoms of any disease.

The following feeding experiments were made: Seven chickens, about five months of age, were fed brains, cords, sciatic nerves, and kidneys from five polyneuritic birds from five different outbreaks. Four of these experiment birds were observed for three months, two were observed for five months, and one was observed for eight months. None of the birds showed any suggestive symptoms during the periods of observation.

Discussion and Summary

In this disease we have a definite affection of the nervous system, which usually produces recognizable gross lesions. The nature of the specific cause is still obscure. However, the histopathology of the nervous tissues strongly suggests that the disease is infectious.

Van der Walle and Winkler-Junius studied a similar, if not identical, disease of chickens in Holland, and reported positive results from feeding and injection experiments. Their results even indicated that the specific cause is a filtrable virus. They frequently mention rather long incubation periods in connection with their inoculation experiments.

In the present study, injection and feeding experiments gave negative results during the periods of observation, which ranged from a few days to eight months in length.

Certain epidemiological features of the disease under farm conditions often suggest a long incubation period. For instance, birds which are obtained as day-old chicks, from an affected flock, often develop the disease when about four to eight months of age; while other birds kept under the same conditions, but originating from an unaffected flock, do not show evidence of the trouble.

An essential relationship between the peculiar iritis and the more strictly nervous affection has not been proved. However, the frequent association of the two conditions in affected flocks and the similarity of the cellular infiltrations tempt one to assume that the two are etiologically identical.

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NEWS ITEMS

The by-laws of the American Society of Clinical Pathologists have been amended, so as to allow veterinarians to hold associate membership.

The Cuban National Veterinary Association, with appropriate ceremonies, presented a handsome flag to the Veterinary Students' Association of the University of Havana on November 23, 1925.

Dr. Abelardo Morejón, of Havana, Cuba, has been appointed Chief of the Municipal Veterinary Service of Santiago de los Cabelleros, Dominican Republic.

STUDIES IN TRANSMISSION OF BACILLARY WHITE DIARRHEA IN INCUBATORS

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INTRODUCTION

A large number of inquiries concerning incubators, as a possible means of disseminating bacillary white diarrhea infection of chicks, led us to study this problem. A thorough search of the literature on the subject revealed no articles dealing with experiments on transmissibility in the incubator. Jones[3] was successful in isolating *S. pullora* from the inner shell membrane of eggs containing dead embryos and suggested incubator infection of chicks by their picking at egg-shells, infected droppings, etc. Rettger, Kirkpatrick and Stoneburn,[4] Durant,[2] Steiner,[5] and many other investigators refer to the possibility of incubator infection. As preventives they advise darkened hatching-trays at the time of hatching and thorough cleaning and disinfection after each hatch.

The continual circulation of chick-down in the forced-air-draught type of machine, at the time of hatching, suggested a possible means of dissemination. If a tray of chicks from an infected flock were hatching in the same incubator as trays from non-infected flocks, would it not be possible for the down from these infected chicks to carry infection to non-infected chicks? This is the problem that we proposed to study.

There are many phases to this problem and before satisfactory conclusions can be made all of these phases must be worked out. In this paper, we wish to dwell upon our preliminary studies of a few of them. These are: (1) the bacteriology of incubators, (2) *S. pullora* infection of chicks through the nostrils, and (3) artificial dissemination of *S. pullora* infection in incubators.

BACTERIOLOGY OF INCUBATORS

We made a qualitative study of the bacteriology of three forced-air-draught type incubators and one sectional type. We then made quantitative studies, and the results of these studies

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will be published in another paper. We found that after each hatch there was a direct relationship between the time of incubation and the number of organisms (in a machine of the forced-air-draught type). After thoroughly cleaning and disinfecting the incubator, we obtained very few organisms. However, as soon as the chicks began to hatch, the number of organisms greatly increased until the chicks were removed. This was not true of the sectional type of machine, where the number remained approximately the same throughout the period of incubation, although at the time of hatching the flora changed to the same as that observed in the forced-air-draught type of machines.

The organisms found to cause this increase at hatching time were of two fecal types. Therefore, if it is possible to get an increase in fecal types of bacteria, it would seem logical to assume that *S. pullora*, which causes bacillary white diarrhea, might also be spread by the chick-down. In our early experiments with a machine which was hatching infected chicks, we were unable to isolate *S. pullora*. Later, in an artificially infected machine, we isolated this organism on three occasions, by exposing agar plates for one minute in various parts of the incubator.

Infection of Chicks Through the Nostrils

Our next step was to determine if chicks might be infected through the nostrils. The following experiment was made to ascertain this. Twenty half-day-old chicks were obtained from a flock free from bacillary white diarrhea. These were divided into three lots. Two lots were treated with pooled, 24-hour, broth cultures of six strains of *S. pullora*, and the third left untreated for controls.

Table I—*Summary of autopsy results on chicks dying from artificial infection through the nostril*

Lot	1		2		3	
History.....	Infected by swabbing nostrils with 24-hr. cultures of *S. pullora*		Infected by injecting one drop of culture into nostrils		Controls	
Total number.....	3		7		11	
	No.	%	No.	%	No.	%
Died—b. w. d.....	2	66.6	7	100.0	1	9.1
Died—other causes.	1	33.3	0	0.0	7	63.6

Lots 1 and 2 received treatment on two successive days. All lots were placed in a brooder and kept separate by means of a board partition. The summary of the results of the experiment is given in table I.

An examination of the records leaves no doubt that the chicks were infected by injecting a pooled culture of *S. pullora* into their nostrils. Nine, or 90 per cent of the chicks which were infected, died from bacillary white diarrhea. One chick of the control lot died from bacillary white diarrhea. As the chicks were from tested stock, this was probably due to infection from the treated lot, which was kept very near the check lot and the same attendant cared for both lots. Overheating is probably the cause of the death of the seven other controls, since the temperature increased several degrees above normal one day during the time the chicks were observed.

Dissemination of Bacillary White Diarrhea in the Incubator

After killing chicks by infection through the nostrils, we decided to try infection in incubators, and the following results of experiments are self-explanatory. A forced-air-draught, 2300-egg-capacity type of machine was used for all phases of this preliminary work, and it was thoroughly cleaned and disinfected after each hatch. Eggs set were from a flock free from bacillary white diarrhea. Controls were not kept in all cases, since records of livability on the 2438 chicks hatched from this flock, over a period of six months, were better than 90 per cent. The average hatchability of the 3505 eggs set from this same flock, over the same period, was 68.4 per cent. Furthermore, they hatched from eggs laid by non-carriers.

Experiment I—Transmission by infected down: The object of this test was to ascertain if chicks hatching in a forced-air-draught incubator could contract bacillary white diarrhea if the organisms were artificially present in that part or another part of the machine. The idea was that the forced circulation of air might carry the organism from one part of the machine to another, on down or any other material that might be present.

Some chick-down was taken from an incubator, sterilized and treated with 24-hour, broth cultures of six strains of *S. pullora*. This down was air-dried and placed in the incubator on the lowest egg-tray (the one closest to the hatching-trays in left compartment of the incubator). There were about 100 chicks

just beginning to hatch in the trays below where the down was placed. There were nine chicks in the opposite compartment of the machine. These were left in the hatching-trays for 38 hours after the down was placed in the machine. The chicks were then put in a brooder and observed. All that died were autopsied and cultures made from the liver, heart, and yolk. The experiment was terminated at the end of 14 days, the remaining chicks were killed and blood samples taken for agglutination tests. Cultures also were made from the liver. By these tests four of the live chicks were found to be carrying the infection. The results of these autopsies are given in table II.

TABLE II—*Summary of results of experiment I. Terminated after 14 days.*

LOT	1		2		3	
History	Hatched in compartment not infected		Hatched in compartment infected		Controls	
Total number	9		108		75	
	No.	%	No.	%	No.	%
Died—b. w. d.	1	11.1	35	32.4	0	0.00
Died—other causes.	0	0.0	5	4.6	1	1.33
Alive at end of experiment	8	88.9	68	62.9	74	98.66
Live chickens suffering from b.w.d. at end of experiment	0		4		Not autopsied	
Total developing b. w. d.	1	11.1	39	36.1	0	0.00

While we were unable to isolate *S. pullora* from the incubator air during this phase, the results obtained from the postmortem examinations indicate that the chicks were artificially infected.

Experiment II—Transmission by down from infected chicks: For this experiment, six strains of *S. pullora* were grown on agar slants for 48 hours and washed off with sterile physiological saline solution. Twelve chicks and eggs were treated with a pool of the above-mentioned cultures. Treatment was given to unhatched chicks after they had pipped, by injecting (through the hole made by pipping) 0.5 cc of the pooled cultures with a syringe and needle. Care was taken to prevent the culture from getting into the mouths of these chicks. The down of ten chicks which had just hatched was also treated by saturating it with the pooled

culture. The idea was to approach what might be natural conditions by placing the organism on the down, allowing it to dry there and possibly be spread through the machine by the moving air.

The infected chicks and the ones exposed in the same compartment were kept separate from those hatching in the non-infected compartment of the machine. They were cared for and fed in the same manner as the chicks of experiment I. The experiment was discontinued after 21 days.

The summary of autopsy results is given in table III.

TABLE III—*Summary of results obtained in experiment II. Terminated after 21 days*

LOT	1		2		3	
History.....	Exposed chicks in infected compartment		Infected chicks		Exposed chicks in non-infected compartment	
Total number.....	30		12		39	
	No.	%	No.	%	No.	%
Died—b. w. d.....	4	13.3	2	16.6	1	2.6
Died—other causes	4	13.3	1	8.4	1	2.6

No controls were kept, since it was thought that previous records of livability of the chicks from the same hens were sufficient to insure the absence of bacillary white diarrhea. All eggs were obtained from tested birds. *S. pullora* was isolated on three agar plates exposed for one minute, in different parts of the incubator at the time of hatching. Forty-two chicks were hatched in the infected compartment of the machine. Six of these died from typical bacillary white diarrhea, as determined by bacteriological examination. Of the 39 chicks hatched in the compartment opposite to the one treated, two died, and of these one died from typical bacillary white diarrhea. No chicks died from bacillary white diarrhea after the thirteenth day from the date of exposure.

Experiment III: In this experiment 135 chicks were divided into groups similar to those in experiment II and were kept under similar conditions.

One hundred twenty-five pipped eggs were removed from the experiment incubator and placed in a small incubator, before being exposed to infection. These were to be used as controls,

but owing to improper management of the incubator, only 25 chicks hatched. Seven of these died within two days, but in no case was *S. pullora* isolated. Also no cultures of *S. pullora* were isolated from the eggs which did not hatch.

The experiment was terminated after 21 days, but the chicks were under observation for over a month. No chicks died from bacillary white diarrhea after the fourteenth day from the time the chicks were artificially infected.

Table IV gives a summary of the results of this experiment.

TABLE IV—*Summary of results obtained in experiment III. Terminated after 21 days*

LOT	HISTORY	CHICKS	DIED—B. W. D.		DIED—OTHER CAUSES	
			No.	%	No.	%
1	Infected before hatching	39	34	87.2	5	12.8
2	Infected after hatching	7	7	100.00	0	00.0
3	Exposed in infected compartment of incubator	30	22	73.3	2	6.6
4	Exposed in non-infected compartment of incubator	55	26	47.3	1	1.8
5	Controls	25	0	00.0	7	28.0

By a comparison of tables II and III, it will be seen that a higher percentage mortality from bacillary white diarrhea was experienced in experiment III than in experiment II. One reason for this may be that earlier artificial infection of chicks was made in experiment III. Only seven chicks were out of the shell when we infected the unhatched chicks in this experiment, while about one-half of the chicks were hatched before the inoculations in experiment II were made. Therefore, by the time the infected down reached the exposed chicks, they were above the age of greatest susceptibility, which is 24 hours (Jones,[3] Brunett[1]). Different strains of *S. pullora* were used for inoculation in experiment III, and it is possible that they were more virulent than those used in experiment II.

Experiment IV: A third experiment, carried out as in experiments II and III, under more nearly field conditions, was deemed advisable, so 20 pipped eggs were inoculated as in previous experiments. These eggs were placed with non-infected eggs

and one non-infected tray was placed in the end of the machine opposite to the infected tray. No chicks had hatched at the time of inoculation. This phase, therefore, differs from experiment II and III in that the exposure in one end might be considered by direct contact. In order to compare field conditions, where two persons might buy the two lots exposed in different manners, lots 1 and 2 were placed in pens separated by about 75 feet of space and fed by two different persons with feed from different containers. In previous experiments, the same person fed all lots, but was careful to feed the less exposed lots first, and took every precaution to prevent infection spreading from pen to pen.

The results of the experiment are summarized in table V.

TABLE V—*Summary of results obtained in experiment IV.*

LOT	HISTORY	CHICKS	DIED		B. W. D.		OTHER CAUSES	
			No.	%	No.	%	No.	%
1	Infected and exposed in same compartment of incubator.....	33	21	63.63	21	63.63	0	0.0
2	Exposed in opposite compartment from lot 1...	46	14	30.44	11	23.91	3	6.5

Remarks: Two others showing symptoms in lot 1. Chicks all appear healthy in lot 2.

The experiment was discontinued after 21 days. No deaths from bacillary white diarrhea occurred in lot 1 after the fourteenth day. In lot 2, one chick died from the disease on the twenty-second day, but none had died, until then, since the twelfth day. No others had died at the end of one month.

No controls were kept in this experiment, for reasons given elsewhere in this paper. Results similar to those reported in previous experiments were obtained, although the percentage of mortality was not as high in either lot as recorded in similar lots in experiment III. These slight variations are to be expected, and may be due to difference in vitality of the chicks, to virulence of the infecting organisms, or to management in the brooding of chicks. The fact that the two lots were cared for under different conditions and by different attendants, with results similar to other experiments, suggests a practical side of the experiment.

The three experiments conducted along the same lines and yielding similar results indicate that it is possible to have incu-

bator infection. The summary of these results is given in table VI.

TABLE VI—*Summary of results obtained from experiments II, III, and IV. All experiments terminated after 21 days.*

DATA	TOTAL CHICKS IN 3 LOTS	DIED		B. W. D.		OTHER CAUSES	
		No.	%	No.	%	No.	%
Chicks hatching in infected compartment of incubator	151	102	67.55	90	59.6	12	7.9
Chicks hatching in non-infected compartment, but exposed to end of incubator	140	43	30.71	38	27.14	5	3.6
Controls	25	7	28.00	0	00.0	7	28.0

In the three experiments, 316 chicks were under observation; 151 of these were either artificially infected or exposed to the artificially infected ones in the same compartment of the incubator; 140 were exposed to the artificially infected chicks, but in the oppostie end of the incubator. The 25 controls were not exposed, and were hatched in a cleaned and disinfected incubator. Higher per cent mortality was observed in chicks exposed in the infected compartment of the machine than in the chicks exposed in the non-infected compartment. This was true also in the experiment where exposure to infected down was the source of disease. Therefore, it seems that there is greater chance of incubator dissemination where normal chicks are in close quarters with the infected ones.

In our studies of the bacteriology of incubators, which will be reported later, we have observed that there is a greater increase in the number of fecal bacteria in the hatching compartment of such incubators than there is in the compartments where no eggs are hatching. Comparing these data with those given in table VI, it would seem that there is a direct relationship between the amount of infection and the amount of infected down carried to the different compartments by the air currents.

DISCUSSION

The results given in this paper suggest a source of bacillary white diarrhea dissemination not reported previously. However, since small numbers are involved, the experiments should be repeated, using larger numbers of chicks and better controls, and naturally infected chicks should be used.

In our preliminary experiments, it was impossible for us to get more than one incubator to work with and the only eggs which were available were some which were being hatched for determination of hatchability. All of these were from birds which were negative to the agglutination test for bacillary white diarrhea and records of hatchability and livability were available on 3505 eggs incubated from this flock during a period of six months previous to the time these studies were started. The livability of the 2438 hatched was over 90 per cent. No outbreaks of bacillary white diarrhea occurred after the flock was tested and reactors removed.

In all the experiments reported above, 529 chicks were used and 111 (20.98%) of these were controls. Only one control died with bacillary white diarrhea, and this occurred in the first experiment to determine if chicks could be infected through the nostrils. In this case the infected chicks were separated only by a loose board partition, and infection could easily be carried by the attendant, by mice or by infected down blown by air. In contrast to this one (0.9%), which died of bacillary white diarrhea in the controls, 170, or 40.68 per cent, of the exposed chicks died with the disease. In all these cases the diagnosis was confirmed by bacteriological examination.

The period of incubation of the disease is from 48 to 72 hours. By a study of results summarized in table II, where the infection depended entirely on the chicks getting some of the down or other infective material, we found that it was four days before the first death occurred in the infected compartment of the machine, and on the ninth day the maximum number of deaths was recorded. After twelve days no more deaths occurred and the chicks appeared normal. With the exception of one instance, no losses from bacillary white diarrhea were experienced in any experiment after the fourteenth day from the date of exposure. In this case the chick died on the twenty-second day.

In a study of the autopsy records, we found that it takes about 24 hours longer to produce infection in chicks, exposed to infective material which is in circulation in the incubator, than where chicks are contaminated by saturation of their bodies with *S. pullora*. Also, chicks infected by saturating their bodies, before they break from the shells, die sooner than those treated from 12 to 24 hours after they are out of the shell. The question of how the disease is produced after such infection is a problem

which still remains to be solved. Probably, when the culture is injected around the chick in the shell, some of it reaches the digestive tract through the mouth or the allantoic circulation. This may account for the earlier peak of mortality in these chicks than in the ones infected in the same manner after they are hatched. The infected down may reach either the digestive tract or the respiratory tract through the mouth or nostrils.

The question of whether the chick-down, pieces of shell, and droppings from naturally infected chicks are a similar source of dissemination yet remains to be settled. There is no doubt that fecal organisms spread throughout the incubators of the forced-air-draught type; and since it has been proved that the infected droppings are one of the chief sources of infection, it is logical to assume that *S. pullora* can be transmitted throughout the machine by the air current. To some extent, this may be true also with other types of machines. We hope to report on this phase of the problem later.

One hatcheryman with whom we have been working states that he has seen typical examples of just such infection, but we have no definite data to prove it. One field example has been reported to us through the kindness of George Robertson, Assistant Dominion Poultry Husbandryman, Canadian Experimental Farms, Ottawa, Canada. He reports as follows:

> "In one instance, we had eggs from a breeder whose flock we afterwards discovered was badly infected with bacillary white diarrhea. I will not give you the results of the whole machine, but simply the results of a line of exhibition Leghorns which we were hatching for Dr. Weaver at the same time.
>
> "The two hatches immediately preceding the hatch referred to gave a chick mortality of 2%. The hatch immediately following the hatch referred to gave 100% viability. In the infected hatch the mortality was 85%. In this connection I might point out that the chicks did not come in contact at all. All the chicks were in wire pedigree baskets and were in separate drawers from the eggs from the infected flock. I might say that the flock from which these White Leghorn eggs came was free of pullorum. We got pullorum from the chicks of the infected flock from the machine, and also from unhatched eggs from the infected flock which were in the machine."

Although we are not ready to affirm that bacillary white diarrhea can be spread in incubators naturally infected, we believe that hatcherymen should be careful to prevent such dissemination by using hatching eggs from tested flocks only. The custom-hatchery problem is one which needs more attention, and every precaution should be taken to insure the owners of custom-hatched chicks that their chicks are not being exposed to infection in the incubator. This can be done (1) by insisting that

clients buy eggs from tested flocks only and (2) by carefully cleaning and disinfecting incubators after each hatch. Custom eggs should not be hatched with eggs intended for hatching commercial chicks, unless it is known that the eggs come from tested flocks.

Conclusion

In conclusion we wish to state:

1. That bacillary white diarrhea was disseminated by artificially infected chick-down, placed in a forced-air-draught-type incubator.

2. That chicks hatching in the compartment opposite to the one in which infected down was spread did not suffer as high a per cent mortality as chicks hatching in the infected compartment.

3. That similar results were obtained by infecting the down of hatching chicks and exposing healthy chicks to the down which might be carried from the infected chicks to the non-infected.

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DR. MILLER TO BUREAU OF DAIRYING

Dr. F. W. Miller has been appointed as veterinarian and physiologist in connection with the breeding research at the five experiment farms in which the Bureau of Dairying is interested. These are at Beltsville, Md.; Ardmore, N. Dak.; Huntley, Mont.; Woodward, Okla.; and Jeanerette, La. He will have supervision of the health of the herds at these places, with special reference to treatment for sterility; and will make anatomical studies with reference to the relation for conformation to milk production, and the physiology of milk production and reproduction in dairy cattle.

Doctor Miller was graduated in 1916 from Ohio State University, where he studied under and was a student assistant of Doctor Sisson. After graduation he went to the Oregon Agricultural College, as instructor in veterinary science and did research work in connection with the control of abortion and sterility. He has had wide experience in veterinary practice among the high-producing herds in the Willamette Valley. During the war he was in the army as a member of the Medical Corps.

B. AERTRYCKE INFECTION IN CANARY BIRDS AND PARROTS*

By F. R. BEAUDETTE, *New Brunswick, N. J.*

New Jersey Agricultural Experiment Station

Epizootics affecting cage birds are not rare, and are usually attended with a high mortality. Such outbreaks occur most frequently in recently imported birds and, as these birds are distributed to other places, new points of infection are established. The writer is informed that four years ago very heavy losses were sustained by bird dealers, which appeared to be due to an infectious agent. A few diseases in cage birds have been investigated and the infecting organism has been diagnosed as *B. paratyphosus B*, at least the organism gave the same fermentation reactions and was agglutinated to the titre limit of an immune *B. paratyphosus B* serum. A few organisms representing a different group have also been isolated from epizootics in cage birds. *B. psittacosis* infection of parrots is well known. Beaudette and Edwards[1] investigated an outbreak in canaries in which the mortality was about 35 per cent of the stock of 200 birds. Nine strains of an organism were isolated from as many specimens and identified as *B. aertrycke*, or *B. pestis caviae*, as termed by some. The identification was based on cultural and biochemic reactions and on agglutinin-absorption tests with known strains of *B. aertrycke*.

The purpose of this contribution is to make a record of a second outbreak of a similar infection, affecting parrots in addition to canaries.

A history of the case has not been obtained, except that the owner stated that about fifty birds had died. A letter addressed to the owner requesting details was not answered and a recent letter was returned.

The canary and small parrot sent for examination had suffered somewhat from postmortem changes, since they were shipped from a place about 150 miles north of New York City. The autopsy showed catarrhal enteritis and congestion of the liver. The lungs were also congested. The serous membrane covering the lower chest wall was reddened. A microcsopic examination of the intestinal contents failed to disclose the presence of parasites.

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Heart-blood and material from the brain of each specimen was streaked on agar plates and incubated at 37° C. At the end of 18 hours of incubation, an abundant growth of a pure culture was obtained on each plate. The organism from the parrot produced acid and gas on dextrose, maltose, mannite, levulose, mannose, sorbitol, arabinose, inositol, rhamnose, xylose, trehalose, galactose, and dulcitol. The amount of gas produced varied from a small bubble, in the case of arabinose, to 75 per cent in the case of trehalose and mannite. Lactose, saccharose, erythritol, raffinose, dextrin and adonitol were not fermented. Glycerol was not attacked in the one test made. The strain from the canary was inoculated into fermentation tubes containing dextrose, maltose, arabinose, inositol, rhamnose, xylose, trehalose, dulcitol, lactose, saccharose and dextrin. All were fermented, with acid and gas production, except the last three mentioned, which were not attacked. Andrade's indicator was used in the fermentation tubes. Neither strain liquified gelatin.

It is apparent from the fermentation reactions that the organism belongs to the paratyphoid-enteritidis group of bacteria. In order to determine the exact identity of the organism, agglutinin-absorption tests were done. In carrying out the absorptions, a heavy bacterial suspension was added to immune serum, so that the final serum dilution was 1-60. The absorption was allowed to take place over night at 37° C. Unabsorbed serum, diluted to 1-60, was also incubated for the same length of time, so that any change brought about by this treatment might be determined.

From the results of the tests, the strains were shown to be agglutinatively the same as *B. aertrycke*. They could not be differentiated from the canary strains isolated by Beaudette and Edwards, nor from strains of *B. aertrycke* isolated during an outbreak of disease in squabs. An immune *B. suipestifer* serum, agglutinating its homologous antigen in a 1-1200 dilution, failed to agglutinate the parrot and canary strains in a 1-120 dilution. This serum continued to agglutinate its homologous antigen after absorption with *B. aertrycke* strains from canaries and pigeons, nor were agglutinins for *B. suipestifer* removed by absorbing the serum with the canary and parrot strain isolated in the present outbreak.

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B. AERTRYCKE AS THE ETIOLOGICAL AGENT IN A DISEASE AFFECTING SQUABS*

By F. R. BEAUDETTE, *New Brunswick, N. J.*
New Jersey Agricultural Station

The outbreak on which this report is made occurred in the vicinity of Camden, New Jersey. The first squab was received for examination, March 9, 1925, and a drop of heart-blood from this specimen streaked on an agar plate gave a pure culture. The bird was about three weeks of age. On March 24, two other squabs were received, the smaller about two weeks of age and the larger less than three weeks old. From the smaller specimen a pure culture was obtained from the heart-blood, liver, kidney, unabsorbed yolk and from an exudate over the brain. The heart-blood and brain of the larger specimen also yielded pure cultures. The plant was visited on April 4 and a fourth squab three weeks of age obtained for examination. Pure cultures were obtained from the liver, heart-blood and brain.

HISTORY AND SYMPTOMS

The owner has been raising squabs for four years and now maintains a loft of 225 pairs. At the time the outbreak started, June 1924, there were 60 pairs of eggs and 54 pairs of squabs. According to the owner, all of the eggs failed to hatch, due either to chilling or infertility and of the 54 pairs of squabs only five or six were marketed. Since that time the disease has constantly claimed its victims, so that several hundred squabs have died. The disease usually attacked the squabs when they were about two weeks of age and death occasionally occurred within two or three days after symptoms developed. According to the owner, it was difficult to keep the squabs in their nests, in spite of the fact that the mothers were good feeders, as evidenced by the full crops of the dead birds. Affected birds displayed a variety of symptoms, indicating mostly a nervous affection. Convulsions were common. Walking in a circle, staggering, twisting of the head and neck, shaking the head, and holding it backwards over the body were symptoms often observed. Occasionally individuals almost completely lost the use of their legs and managed to get around only by the use of their wings. Very young squabs often threw themselves from the nest.

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A yellow color of the eye was noted in some cases, while the eyeball appeared white and glassy in others. Such changes were usually unilateral.

Diarrhea was often observed and the droppings were yellow, white and occasionally grass green in color.

Occasionally a week would pass without an appearance of the disease but it always returned in spite of the fact that the cotes were disinfected regularly with equal parts of kerosene and crude carbolic acid. The disease did not seem to affect a large number of squabs at one time, but claimed its victims regularly. There appeared to be a slight increase in the incidence of the disease during the warmer months. On March 23, the owner removed 23 squabs, of which only fifteen were marketable and eight were culls, probably affected with the disease. There were three dead ones removed on this occasion. This seemed to be typical of each lot removed. Ordinarily the owner secured trom six to eight pairs of squabs from each pair of breeders, but after the disease started the number was greatly reduced. Old birds never contracted the malady, according to the owner.

A visit made to the plant showed that the birds were housed in three compartments. Nests extended from the floor to the ceiling on three sides of each compartment. The birds appeared to be overcrowded, since the owner had made it a practice to keep them out of the flies during wet weather and such was the case when the visit was made. The houses appeared insanitary and puddles of water stood in the yard. The owner was of the opinion that lime water given as drinking water seemed to check the disease. At least, since its use, he had been able to market a larger number of birds.

Autopsy Notes

A congestion of the lungs and kidneys was noted in the first squab autopsied. The liver was congested and enlarged. The intestines contained a catarrhal exudate and reddening of the mucosa was observed. The blood-vessels of the intestines were injected. No hemorrhages were found on the heart.

In the smaller specimen of the second lot examined the crop was filled. The blood-vessels surrounding the proventriculus were injected. The gizzard was normal, though the intestines were catarrhal. The blood-vessels of the intestines were injected and the mucosa showed occasional hemorrhages. The liver was enlarged and congested, though fairly firm in consistency. The

pancreas of this specimen showed pin-head-sized white or grayish nodules throughout. The kidneys were soft and pale and the ureters were slightly enlarged. In the lower anterior portion of the right lung a caseous nodule was found. A bit of unabsorbed yolk, about the size of a pea, was found attached to the intestine by a slender stalk. The heart showed no hemorrhages. On opening the cranial cavity an exudate was found covering a portion of the right cerebrum and cerebellum. The larger squab of this lot showed practically the same changes, except that nodules were not found in the pancreas, but instead these small nodules were scattered throughout the length of the small intestine, buried in the wall though visible through the serous surface. A caseous nodule was found in the lung on the same side and in about the same place as in the preceding specimen. There was no exudate over the brain. An unabsorbed yolk sac was present.

The lesions found in the fourth specimen were about the same as those described above for the first specimen.

Bacteriological Study

Agar plates streaked with heart-blood or with material from the organs showed an abundant growth at the end of 18 hours of incubation at 37° C. After 48 hours the colonies did not enlarge much.

The organism was found to produce acid and gas in dextrose, maltose, glycerol, mannite, levulose, mannose, sorbitol, arabinose, inositol, rhamnose, xylose, trehalose, galactose, and dulcitol. Neither acid nor gas was produced in lactose, saccharose, melezitose, erythritol, raffinose, dextrin, inulin, soluble starch, salicin, adonitol, or amygdalin. Fermentation of maltose and inositol was delayed until 72 hours of incubation and glycerol and xylose were not fermented until 120 hours.

Growth occurred along the line of puncture in a gelatin tube and spread over the surface somewhat. There was no liquefaction.

The fermentation reactions indicate that the organism belongs to the paratyphoid-enteritidis group, and its ability to attack xylose, trehalose and inositol shows it to be rather closely related to *B. schottmulleri*. These reactions exclude the possibility of its being *B. paratyphosum A*. In order to determine its identity, agglutinin-absorption tests were made.

Serological Tests

In some of the absorption tests done in the beginning, a heavy bacterial suspension was added to the immune serum to be absorbed, so that the final serum dilution was 1-60. The absorption was allowed to take place during three or four hours of incubation at 37° C. The tubes were then centrifugalized and the clear, supernatant fluid removed with the aid of a capillary pipette. The proportion between sediment and fluid was about 1 to 7. In later experiments, the same dilution of serum was used with about the same bacterial suspension, though the tubes were incubated overnight. It was found that complete sedimentation took place in tubes where the serum was related to the absorbing antigen and centrifugalization was not necessary.

The absorption tests with an immune *B. suipestifer* serum showed the organism to be agglutinatively different from this type.

Tests with immune B. aertrycke serum: This serum, absorbed with its own antigen, failed to agglutinate the pigeon strains under study. When the serum was absorbed with a *B. aertrycke* strain from canaries, the agglutinins were removed for *B. aertrycke*, pigeon typhus and for the absorbing antigen. When absorbed by the pigeon-typhus strain, the serum failed to clump its absorbing antigen as well as *B. aertrycke*, *B. aertrycke* from canaries and the pigeon strains under study. When *B. anatum** was used to absorb the serum, there was no reduction of agglutinins for *B. aertrycke*, *B. aertrycke* from canaries, pigeon typhus and the pigeon strains under study. Likewise, saturation of the serum with *B. schottmulleri* failed to remove the agglutinins for these types.

Tests made with serum immune to B. aertrycke from canaries (L-271-1): The results of the absorption tests with this serum were essentially the same as those obtained in the preceding case.

In table I, where the serum was absorbed with pigeon typhus, it is evident that the absorbing antigen was not heavy enough, as agglutination continues to occur in the lower dilutions, except in the case of *B. anatum*, which is agglutinatively different.

Absorption tests with serum immune to pigeon L-660: This serum, unabsorbed, caused complete agglutination of its homologous antigen, *B. aertrycke*, *B. aertrycke* from canaries and the

*Rettger and Scoville have isolated a type of *B. anatum* from ducklings which is identical with *B. aertrycke*, but the type used in these experiments is agglutinatively different from *B. aertrycke*. See reference 6 below.

TABLE I.—*Showing results of absorption tests with immune B. aertrycke serum. (Immunizing strain, L-271-1, from canary bird.)*

ABSORBED BY	AGGLUTINATES	SERUM DILUTIONS						
		1-120	1-240	1-600	1-1200	1-2400	1-4800	CONTROL
Unabsorbed	Pigeon L-660	++++	++++	++++	++++	++++	++	○
Pigeon L-660	Pigeon L-660	+	○	○	○	○	○	○
"	*B. aertrycke* APT 2	+	○	○	○	○	○	○
"	*B. aertrycke*, canary	++	○	○	○	○	○	○
"	*B. anatum* C-1	○	○	○	○	○	○	○
"	Pigeon typhus	○	○	○	○	○	○	○
Unabsorbed	*B. aertrycke* APT 2	++++	++++	++++	++++	++++	+++	○
B. aertrycke APT 2	*B. aertrycke* APT 2	++	++	○	○	○	○	○
"	Pigeon L-660	+	○	○	○	○	○	○
"	*B. aertrycke*, canary	+++	+	○	○	○	○	○
"	*B. anatum* C-1	○	○	○	○	○	○	○
"	Pigeon typhus	○	○	○	○	○	○	○
Unabsorbed	*B. aertrycke*, canary	++++	++++	++++	++++	++++	++++	○
B. aert. canary	*B. aertrycke*, canary	+	○	○	○	○	○	○
"	Pigeon L-660	+	○	○	○	○	○	○
"	*B. aertrycke* APT 2	+	○	○	○	○	○	○
"	*B. anatum* C-1	○	○	○	○	○	○	○
"	Pigeon typhus	○	○	○	○	○	○	○
Unabsorbed	*B. anatum* C-1	+++	○	○	○	○	○	○
B. anatum C-1	*B. anatum* C-1	○	○	○	○	○	○	○
"	Pigeon L-660	++++	++++	++++	++++	++++	++	○
"	*B. aertrycke* APT 2	++++	++++	++++	++++	++++	+++	○
"	*B. aertrycke*, canary	++++	++++	++++	++++	++++	++++	○
"	Pigeon typhus	++++	++++	++++	++++	++++	++	○
Unabsorbed	Pigeon typhus	++++	++++	++++	++++	++++	++	○
Pigeon typhus	Pigeon typhus	++++	++	++	+	○	○	○
"	Pigeon L-660	++++	+++	+	+	○	○	○
"	*B. aertrycke* APT 2	++++	+++	++	+	○	○	○
"	*B. aertrycke*, canary	++++	++++	++	+	○	○	○
"	*B. anatum* C-1	○	○	○	○	○	○	○

Symbols: ++++, complete agglutination; +++, marked agglutination; ++, partial agglutination; +, trace of agglutination; ○, no agglutination.

pigeon typhus strain, in a dilution of 1-2400, and partial agglutination in a dilution of 1-4800. When absorbed with any one of these strains, the serum failed to clump the absorbing antigen as well as the remaining ones. The unabsorbed serum caused only a slight clumping of *B. anatum* in a 1-120 dilution. The serum absorbed with *B. anatum* continued to agglutinate *B. aertrycke*, *B. aertrycke* from canaries, pigeon typhus and the pigeon strain under study, but failed to cause clumping of *B. anatum* in the 1-120 dilution.

Absorption tests with serum immune to B. anatum C-1: The unabsorbed serum agglutinated *B. anatum* but failed to agglutinate *B. aertrycke*, *B. aertrycke* from canaries, pigeon typhus and the pigeon strain under study. Absorption of this serum with any of the unagglutinated strains failed to alter the agglutinins for *B. anatum*.

Absorption tests with serum immune to pigeon typhus: When absorbed by its own antigen, the serum failed to agglutinate the pigeon strains isolated from the outbreak under study, as well as *B. aertrycke*, *B. aertrycke* from canaries and the immunizing strain. Likewise, saturation of the serum with the pigeon strain L-660 removed the agglutinins for pigeon typhus, *B. aertrycke* and the canary strain.

Source of Cultures

B. aertrycke (APT. 2)—Stock strain from Dr. Rettger.

B. aertrycke (L-271-1) Canary 1—Isolated from epizootic in canary birds.[1]

B. anatum (C-1)—Isolated by Dr. Rettger from duckling.

Pigeon typhus—From Dr. Th. Smith, who in turn obtained it from Washington, D. C., several years ago. Probably isolated by Mohler in 1904.

Pigeon (L-660)—Isolated from first squab examined in present outbreak.

B. suipestifer (17)—Stock culture from Dr. Th. Smith.

B. paratyphosum B (12)—From Dr. I. E. Newsom, who in turn obtained it from Dr. E. O. Jordan.

Similar Diseases of Pigeons Investigated by Others

Moore[2] investigated a disease in pigeons in 1894 and points out its prevalence in young birds. Old pigeons occasionally contracted the disease, however, though they manifested a more chronic form. The symptoms displayed by the old birds were similar to the ones described above as seen in the present

O, no agglutination.

outbreak. Marked emaciation was noted by the owner. Moore was able to isolate an organism from the heart-blood and liver of a squab six weeks of age and from an exudate over the brain of an adult bird. However, in the latter specimen he was unable to obtain a culture from the heart-blood. The organism did not liquefy gelatin, but produced acid and gas in dextrose. Lactose and saccharose were not fermented. Moore states that the organism is a variety of the hog cholera bacillus, but differs from it in some respects.

The Twenty-first Annual Report of the Chief of the Bureau of Animal Industry for 1904 records an infectious enteritis of pigeons. It is stated that an organism of the enteritidis group was isolated from the brain, heart, liver, spleen and kidney of affected specimens. Mohler[3] states that seven outbreaks were investigated in which both squabs and adults were affected. The mortality was about 25 per cent. Four of the outbreaks occurred in Washington, D. C., one in St. Louis, Missouri, Sullivan County, Indiana, and in Atlantic County, New Jersey. A large number of birds were never affected at one time, but the disease continued to take its victims until 50 per cent of the pigeons succumbed in one loft. Diarrhea was a constant symptom. There was a focal necrosis in the liver and spleen, a marked inflammatory condition of the proventriculus and numerous ulcerations of the mucosa of the duodenum and large intestine down to the cloaca, resembling those observed in chronic hog cholera.

The pigeon typhus organism used in the present study was probably isolated by Mohler from one of these outbreaks.

In 1914, Zingle[4] reports on an epizootic among pigeons in Strasburg. Emaciation was an outstanding symptom. The intestines were hemorrhagic and the kidneys enlarged and of a grayish-yellow color. From the blood and organs an organism was isolated which could not be differentiated from *B. paratyphosum B* morphologically, culturally or serologically. The author considers the possibility of mixed infection.

McGowan (1915)[5] considers the organism isolated by him from two pigeons as an intensified type of the fowl cholera germ. The strain isolated from the heart-blood and spleen of one pigeon produced acid and gas on glucose, mannite, dextrin, dulcite, galactose, maltose and sorbite, but neither in cane sugar, adonite, lactose, inulin, nor salicin. The strain from the heart-blood of a second case was similar, except that it failed

to ferment mannite, produced indol and formed acid and doubtful coagulation in litmus milk. The organism from the spleen of the same specimen was markedly different.

Discussion

It is evident from the results obtained in the agglutinin-absorption tests that the organism under study cannot be differentiated from *B. aertrycke*. The identity of the organism with the pigeon typhus strain is also apparent. The description of the disease left by Moore is very similar to the findings in the present outbreak, and in all probability the causative organisms are identical. Some of the strains studied by McGowan seem to bear some relationship to this type of organism.

Moore brings out the point that the disease investigated by him is known as "megrims" to pigeon breeders. From the literature cited, it is evident that the infection is not uncommon. Attention is called to the fact that the infection has been located in three counties in New Jersey. Moore first observed it in 1894, in Vineland, Cumberland County. Mohler located the infection in Atlantic County, in 1904, and the present outbreak was found in Camden County, in 1925. All three counties are located in the southern part of the State.

The isolation of the organism from an unabsorbed yolk sac suggests the possiblity of infection being transmitted through the egg. This is further suggested by the fact that in the present outbreak, and in the ones reported by Moore and Mohler, the disease persisted in spite of disinfection and sanitary precautions. As is well known, white diarrhea cannot be prevented in baby chicks by sanitary measures when the infection is introduced by way of the egg. The appearance of the disease in very young squabs also makes transmission through the egg seem plausible, though it must not be overlooked that the mode of feeding in pigeons may account for the appearance of the disease in early life.

The ability of infections of this type to localize in various parts of the body is well known. The localization of the white diarrhea organism has already been mentioned. Rettger and Scoville[6] have suggested possible transmission of infection through the egg in the case of *B. anatum*, Beaudette[7] has reported on evidence in favor of *B. sanguinarium* infection being transmitted through the egg. It was pointed out that Moore was unable to isolate the organism from the heart-blood

of an adult pigeon, but the organism was isolated from an exudate over the brain, where it had apparently become localized.

SUMMARY

An organism which cannot be differentiated from *B. aertrycke* was isolated from the heart-blood, liver, kidney, brain and unabsorbed yolk sac of squabs dying of a disease known as "megrims."

The fermentation reactions of the organism are identical with those of *B. paratyphosum B.* By means of agglutinin-absorption tests, the organism was shown to be agglutinatively different from *B. paratyphosum B* and *B. suipestifer*. The organism was found to be agglutinatively identical with *B. aertrycke*.

The organism was found to be the same as a pigeon typhus strain, isolated probably by Mohler. The similarity of the outbreak with those reported by others indicates that this disease is not uncommon.

Transmission of the infection through the egg seems possible, since the organism was isolated from an unabsorbed yolk, the disease attacks very young squabs. and because sanitary precautions fail to prevent its constant appearance.

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BUREAU TRANSFERS

Dr. F. Weaver (McK. '10), from Baton Rouge, La., to Chicago, Ill., on meat inspection.

Dr. Cecil D. Emerick, from Little Rock, Ark., to Chicago, Ill., on meat inspection.

Dr. Calvin S. Evans (Chi. '03), from Raleigh, N. C., to Buffalo, N. Y. on meat inspection.

Dr. Leo E. Davis (O. S. U. '11), from hog cholera control to meat inspection, Columbus, Ohio.

NEW EDITION OF HUTYRA AND MAREK

Announcement is made elsewhere in this issue of the JOURNAL of the publication of a new edition of Hutyra and Marek, in three volumes. A very attractive offer is made by Alexander Eger to those who order a set in advance. Veterinarians taking advantage of this offer will save about ten dollars by so doing.

PLAN FOR HANDLING AVIAN TUBERCULOSIS

By T. E. MUNCE, *Harrisburg, Pa.*

Pennsylvania Bureau of Animal Industry

A few years ago the Pennsylvania Bureau of Animal Industry found that many flocks of poultry in one county of Pennsulvania were affected with tuberculosis. The mortality in these flocks was high and the owners were annoyed not only at their inability to maintain healthy flocks, but also at the monetary loss sustained. The disease had been increasing for the past few years in all affected flocks, while some of the flocks had been experiencing losses for years. The owners were anxious to do something regarding this disease. Plans were made to undertake work in some of these flocks for the purpose of becoming better acquainted with the tuberculin test as well as to study the prevention and eradication of this disease.

Considerable work has been done in these flocks. From the results obtained and from our experience with tuberculosis the following plan was prepared and is now being used by the Pennsylvania Bureau of Animal Industry for handling avian tuberculosis on a flock basis.

PENNSYLVANIA DEPARTMENT OF AGRICULTURE
Bureau of Animal Industry

PLAN FOR HANDLING AVIAN TUBERCULOSIS ON A FLOCK BASIS

1. Determine the extent of the infection by history, tuberculin test and autopsy.
2. Removal and slaughter of diseased fowls, under official supervision. In extensively infected flocks the exposed fowls should also be slaughtered.
3. Clean thoroughly and disinfect premises under official supervision.
4. Flocks from which diseased fowls have been removed, but where the entire flock has not been slaughtered, should be given a physical examination and all fowls in good condition tuberculin tested and should be retested in 60 to 90 days.
5. In the event of reactors being disclosed to the 60 to 90 day retest, the flock should be again tested in 6 months.
6. After a negative test and all fowls are in good physical condition the flock should be tuberculin tested annually.
7. All diseased fowls should be killed under official supervision.
8. Droppings should be disposed of so as not to come in contact with poultry.
9. Cleanliness should be maintained and frequent disinfection used.
10. Fowls other than baby chicks should be tuberculin tested before being added.
11. Uncooked table scraps and similar refuse should be discontinued as feed.

*Contribution from the Pennsylvania Bureau of Animal Industry No. 22.

12. Consistent effort should be made to exterminate carriers such as rats, mice and pigeons
13. In the prevention and eradication of avian tuberculosis, attention should be given the tuberculosis problem in all kinds of associated live stock, especially swine tuberculosis.
14. Owners will be expected to practice careful breeding, correct feeding, proper flock management, principles of sanitation and to use preventive measures toward other fowl diseases.
15. The following certificate is issued to owners who will agree to follow the above plan and whose fowls have passed two annual negative tuberculin tests

AGREEMENT OF OWNER WITH THE BUREAU OF ANIMAL INDUSTRY, PENNSYLVANIA DEPARTMENT OF ARGICULTURE

I hereby agree to the terms of the above plan for the prevention, repression and eradication of avian tuberculosis. I further agree to observe and fulfill the foregoing requirements and failure on my part shall be sufficient cause for cancellation of this agreement.

My flock consists of ....., ...
Number Breed

Date .....192.... Owner .....

Witness ..... Address .....

CERTIFICATE OF INSPECTION AND TEST FOR AVIAN TUBERCULOSIS

No..... Issued .....192...

This is to certify that the entire flock of .....,
Owner

....., consisting of ..... fowls, has
Address Number

undergone inspection; has passed the required number of tuberculin tests; and that the owner has complied with the requirements prescribed by the Pennsylvania Bureau of Animal Industry for the prevention, repression and eradication of avian tuberculosis.

This certificate is good for one (1) year from date unless revoked.

.....192...
Date

.....
State Veterinarian.

If a flock is diseased and a diagnosis of tuberculosis is established, in order to give the proper advice it is necessary to determine the extent of the infection. Aids in such a determination are the history, tuberculin test and autopsy. Should a history be given of an unprofitable flock, with some losses by death, the oldest birds showing the advanced symptoms and most deaths, birds going light, anemia, and many individuals going lame, it can be inferred that the flock is badly infected. Autopsy of several birds showing advanced lesions and generalized cases would also indicate extensive infection. The accurate method of determining the exact extent is by means of the tuberculin test.

All diseased birds should be slaughtered under official supervision to insure the proper disposition of the meat, as well as to aid in giving proper advice regarding the flock. If infection is extensive, the most practical, economical and efficient method of eradicating avian tuberculosis is by the slaughter of the entire flock. This is the shortest method and is by far the most complete.

After such a procedure, or even where the birds are tested and the reactors are removed, the premises should be disinfected under official supervision. The average flock-owner does not understand the insidious nature of disease-producing organisms and does not appreciate the necessity for thorough cleaning. Where the entire flock cannot be slaughtered, all birds should be given a physical examination, since badly affected cases many times fail to react to the tuberculin test, as is true in bovine tuberculosis. Those fowls in good physical condition should be carefully tuberculin tested, reactors removed, premises cleaned and disinfected under official supervision and retested in 60 to 90 days. If further reactors are disclosed, the flock should be again tested. When negative it is believed that an annual test will be sufficient to guard against future inroads of tuberculosis.

Droppings should be disposed of so as not to come in contact with poultry. It is known that many tuberculous fowls have tuberculosis of the intestines, with nodules discharging large numbers of tubercle bacilli into the feces. This contaminates a premise and everything about it and is believed to be the common method of transmission.

Cleanliness must be maintained to minimize the amount of infection and disinfection used to destroy the organisms. These measures are known to be necessary in all kinds of disease control.

Lesions of tuberculosis have not been found in baby chicks and since they cannot be tested practically, it is believed they can be added to a flock without danger of introducing tuberculosis. Mature fowls should be tested in order to determine if free before being added to a flock.

Uncooked table scraps may contain the offal from infected fowls, since it is common practice to dispose of the offal by placing it in the garbage. Rats and mice can be infected artificially and pigeons are commonly known to be affected, so must be considered dangerous as a means of spreading tuberculosis. By habits of association with the farm flock, the rats and mice are usually present about poultry premises as well as often living

in the feed-room. Pigeons frequently feed with the barnyard fowls and also have a habit of visiting and sojourning at different farmyards and premises.

Experimental work has shown conclusively that swine become infected naturally by association with tuberculous fowls and it is believed they in turn may transmit the disease to poultry. Chickens and swine frequently associate closely.

In any disease prevention control or eradication work, due regard should be paid to careful breeding, correct feeding and proper flock-management, together with good sanitation and preventive measures against other disease.

A formal agreement between an owner and the agency working for disease prevention and eradication is necessary to be legal, as well as to obligate and impress the owner as to the necessity for his part in the work.

When such an agreement has been fulfilled in a satisfactory manner and the desired result obtained, a certificate is issued.

SCIENTIST APPOINTED TO BUREAU OF DAIRYING

George W. Hervey has been appointed as biometrician in the Bureau of Dairying, to assist in the study of the underlying principles governing the heredity of milk and butterfat-producing ability in dairy cattle. There is a vast amount of data in the records of production of the various breeds that will have an important bearing upon breeding research, when studied by biometrical methods. Research of this character, which requires an extensive application of higher mathematics as well as a broad knowledge of genetics, will be undertaken by Mr. Hervey.

Mr. Hervey was graduated from Rutgers College in 1915. He was instructor in animal husbandry in the University of Arkansas and for several years was instructor and extension professor in poultry husbandry in the University of Missouri, where, at the same time, he secured the degree of M. A. He was later assistant professor and acting professor in charge of poultry husbandry at Rutgers College and research specialist in the New Jersey Agricultural Experiment Station. He studied for his doctorate at Harvard University, majoring in genetics and biometrical methods. During the war he served in the field artillery and saw active service in the battles of St. Mihiel and Argonne Forest and was later in the Army of Occupation in Germany.

CLINICAL AND CASE REPORTS

(Practitioners and others are invited to contribute to this department reports of unusual and interesting cases which may be helpful to others in the profession.)

AN OUTBREAK OF FOWL CHOLERA*

By A. K. Gomez

College of Veterinary Science, Los Banos, Laguna, P. I.

Introduction

The poultry industry in the Philippines is rife with great possibilities. It is essential, therefore, that we should not only learn all that pertains to the breeding and rearing of the avian species, but we should also know the diseases that affect them and the methods of combating and preventing these epizootics.

In the United States, poultry diseases constitute a big problem in veterinary research work. Specialists in this particular line form a group of scientific "chicken pathologists." Recently, veterinary journals have devoted whole issues to the discussions of poultry ailments.

In the Philippines, very little, if any, attention is given to this important field of investigation. For the purpose of awakening more interest in chicken diseases here, this study of a recent outbreak of fowl cholera is presented.

Autopsies

From December 6, 1924, to March 26, 1925, there occurred heavy losses among the chickens, ducks, and turkeys of the Poultry Division of the College of Agriculture. The dead birds were sent to the College of Veterinary Science for autopsy. The majority of these cases showed lesions pathognomonic of fowl cholera. Only a few of these autopsy examinations are given here, all of them being on file in the Department of Pathology and Bacteriology of the College of Veterinary Science.

On February 19, 1925, dead Cantonese hens B231, B347, B243, all from pen 9, which registered a heavy mortality, were sent to the College of Veterinary Science for postmortem examination.

The following lesions were found:

1. Cantonese hen B231: Comb and wattles bluish black in color; rigor mortis, marked; heart covered with a fibrino-purulent exudate; pericardium adherent to the heart muscles; left lung hepatized and grey in appearance. There were extensive hemorrhages on the right side of the peritoneal cavity; the spleen showed prominence of the malpighian corpuscles; the liver was light chocolate-brown in color and was riddled with pin-point hemorrhages throughout. The mucous membrane of the duodenum was petechiated, and its contents consisted of a pasty, slate-grey mass, admixed with minute blood-clots.

*Read at the 13th scientific meeting of the Los Baños Biological Club.

Microscopic examination of blood-smears from the heart, liver, and spleen, stained with 1-10 dilution of carbol-fuchsin for three minutes, revealed the presence of bipolar organisms.

2. Cantonese hen B347: Marked rigor mortis; comb and wattles, cyanotic; numerous petechiae along the coronary region of the heart; a gelatinous exudate tinged with blood in the pericardium; liver apparently normal and the mucous membrane of the duodenum was reddened.

Microscopic examination of blood-smears from the heart, liver, and spleen showed presence of bipolar organisms.

3. Cantonese hen B243: Comb and wattles, cyanotic; rigor mortis, pronounced. Heart apparently normal; liver had a cooked appearance and was spotted with numerous greyish-white dots of degeneration, varying in size from a pin-point to a pin-head. The mucous membrane of the small intestine was reddened.

Microscopic examination of smears made from the heart-blood, liver, and spleen revealed the presence of bipolar organisms.

On March 14, 1925, a large gobbler was sent to the College for autopsy. This turkey was found dead at the roosting-place, without having shown any previous signs of disease.

The postmortem findings were as follows: rigor mortis, marked; the fleshy portion and wattles, cyanotic; heart, petechiated; coronary arteries, engorged; a sero-sanguinous transudate (30 cc) in the pericardium; liver, pale chocolate-brown and riddled with greyish-white foci of degeneration, varying in size from a pin-point to a pin-head; mesentery, petechiated and mesenteric arteries injected; visceral surface of the gizzard showed numerous petechiae; lungs edematous.

Microscopic examination of the heart-blood revealed the presence of bipolar organisms.

On March 18, 1925, a Rhode Island Red hen (B680) was received at the laboratory of the College for autopsy. The findings were as follows: rigor mortis, marked; crop, full; pericardium contained about 5 cc of sero-sanguinous transudate; petechiae at the coronary region of heart. A large, red coagulum was present in the peritoneal cavity, covering the entire liver. This organ showed numerous greyish-white dots of degeneration throughout, and hemorrhages were found in the membranes of four large ova; the arteries of the oviduct, markedly engorged; the fat on the visceral surface of the gizzard was petechiated.

Microscopic examination of the heart-blood revealed presence of bipolar organisms.

On March 25, 1925, another Cantonese hen (B68) from pen 9 was received for postmortem examination. The findings were reported as follows: heart, petechiated; multiple focal necrosis of the liver (this organ had a pale, chocolate-brown color and was friable); congestion of the mucous membrane of the intestines.

Microscopic examination of the blood revealed few bipolar organisms.

On March 26, 1925, dead Cantonese hen (B59) was sent to the laboratory for autopsy. The findings were as follows: comb, cyanotic; liver, pale chocolate-brown, friable and riddled with numerous greyish-white areas of degeneration, varying in size from that of a pin-point to that of a pin-head; a large, red coagulum was found adherent on the left middle lobe; visceral surface of the gizzard, petechiated; mesenteric vessels, congested. On several ova, there were present minute, white spots in the yolk, from which pure cultures of bipolar organisms were recovered. These ova were also covered with large hemorrhages. The kidneys were enlarged and reddened. Spleen showed no change. The pericardium contained a gelatinous exudate, and the heart was petechiated. Lungs, edematous.

Microscopic examination of smears from the heart-blood, liver, and lungs revealed the presence of numerous bipolar organisms.

The Causative Agent

Pure cultures of *Pasteurella avicida* were recovered from the heart, liver, and spleen of the dead fowls submitted for autopsy.

They were cultivated in artificial media and their biological characteristics studied. Organisms from a 24-hour culture on agar slants or from tissues were readliy stained with ordinary aniline stains, but were found to stain best with a 1-10 dilution of carbol-fuchsin, allowed to act on a fixed smear for 3 to 5 minutes. They did not retain the stain with Gram's method. Grow well at 37.5° C. Under the microscope, the organisms appeared as minute rods, measuring on the average 0.84 μ by 0.42 μ. They showed polar staining.

Experimental Inoculations

The disease was easily transmitted to susceptible fowls by injections of the culture or fluids from chickens dead of the disease.

On February 19, 1925, two healthy chickens were inoculated with 0.5 cc of pericardial fluid from Cantonese hen B347, one intraperitoneally and the other intramuscularly. Both chickens died in 14 and 18 hours respectively. On autopsy, they showed identical lesions, consisting of petechiated heart and liver, and congestion of the mucous membrane of the intestines. The one injected in the breast muscle showed a straw-yellow, necrotic tract at the path of the inoculation needle. Microscopic examination of the heart-blood showed the presence of bipolar organisms.

From one of these chickens (the one that died 18 hours later), which was autopsied immediately after dying, about 0.5 cc of the heart-blood was diluted with about 30 cc of sterile salt solution and placed in a sterile bottle. One-half cc of this solution was injected into the breast muscle of a healthy chicken, and the remainder was passed through a Berkefeld filter, and about 5 cc of the filtrate was inoculated intramuscularly into another healthy chicken.

The chicken injected with the filtrate survived, while the one injected with the diluted blood died.

On March 20, 1925, two healthy chickens, one a small cockerel (E1) and another a hen (E2), both Cantonese, were placed in an infected cage and fed with infected feed.

On March 25, 1925, chicken E1 was found dead in the cage, without showing any previous symptoms. Postmortem lesions were as follows: fibrino-purulent pericarditis; heart, petechiated; peritoneum, dry; liver, dark brown in color, with engorgement of the central veins; mucous membrane of the small intestines, reddened; surface of the gizzard showed few petechiae.

Microscopic examination of the heart-blood showed presence of bipolar organisms. Pure cultures of *P. avicida* were recovered from the heart-blood, liver, and spleen.

Cantonese hen E2 was very sick this day, and the following symptoms were observed: somnolence; sits continuously in a corner of the cage with feathers ruffled; watery, yellowish-white feces; anorexia; when disturbed, is nervous.

On March 27, 1925, this hen was killed, as she was very weak, and autopsy was performed immediately.

The lesions shown were as follows: fibrinous pericarditis; petechiae along the coronary region of the heart; multiple focal necrosis of the liver.

Microscopic examination of the heart-blood showed presence of bipolar organisms.

The liver, heart, and spleen of this hen were taken, washed with sterile salt solution and immersed in a beaker, containing 1 per cent solution of phenol, overnight. The following morning, these organs were washed in sterile salt solution, cut into small pieces by means of a sterile pair of scissors, and triturated in a sterile mortar and pestle. The mass was then mixed with chicken-feed, and this was fed to four native healthy chickens (E3, E4, E5, and E6). Two others of the same lot were kept in a separate cage as controls.

Twenty-four hours after feeding, chicken E6 was dead and E4 very sick, while E3 and E5 showed nothing abnormal in their behavior.

The lesions found in E6 were as follows: comb and wattles, cyanotic; sero-sanguinous transudate in the pericardium; petechiae of the heart; edema of the lungs; congestion of the mucous membrane of the small intestines.

Microscopic examination of the heart-blood showed presence of bipolar organisms.

The following symptoms were observed in chicken E4: inappetence; continuously lying down in a corner of the cage; somnolence; temperature, 42.6° C. This chicken was found dead in the cage on April 1, 1925, six days after feeding with the infected feed. On postmortem, it showed: emaciation; dry subcutis; petechiated heart; ventricles filled with dark coagulum; liver friable and passively congested.

On April 3, 1925, chicken E3 was found dead in the afternoon, having shown no visible symptoms. The lesions on autopsy were as follows: petechiae of the heart; congestion of the coronary arteries; edema of the lungs; petechiated liver; congestion of the mesenteric vessels; intestinal contents, slate-grey and admixed with blood clots; petechiae on the visceral surface of the gizzard.

Microscopic examination of smears from the heart-blood, liver, and spleen revealed presence of bipolar organisms. Pure cultures of *P. avicida* were recovered from the heart-blood.

The controls and chicken E5 survived.

On May 6, 1925, a 24-hour agar culture of the organism was suspended in sterile salt solution, and about 2 cc of this was injected into the pectoral muscle of a native brown pullet (E7) at 10:00 a. m. This pullet was found dead at 6:25 p. m. the same day.

Postmortem findings were as follows: comb, cyanotic; rigor mortis, pronounced; heart had a cooked appearance and a few petechiae along the coronary region; a gelatinous exudate was present in the pericardium; left lung, edematous; sero-sanguinous exudate in the pleural cavity. The mesenteric vessels were engorged, and contents of the duodenum was a slate-grey, pasty mass, admixed with blood-clots. There was a sero-sanguinous transudate in the peritoneal cavity. The cloaca was markedly reddened, and the feces were watery. Pectoral muscles showed, at the point of injection, a whitish, necrotic tract, with arborization of the surrounding arterioles. There was present a straw-yellow exudate. Liver was pale red in color; spleen, dark brown; and kidneys, swollen and reddened.

Microscopic examination of the heart-blood showed presence of bipolar organisms.

Pure culture of *P. avicida* was recovered from the heart-blood.

May 12, 1925, a native brown pullet (E8) was injected with 0.5 cc of this culture subcutaneously.

Chicken E8 showed the following symptoms on May 14, 1925: somnolence; anorexia; temperature, 44° C. Red blood corpuscles, 2,984,000 per cc; white cells, 32,000 per cc.

May 15, 1925: same symptoms; blood counts showed red cells, 2,392,000 per cc, and white cells, 25,000 per cc.

May 16, 1925, the chicken was found dead in the cage. Autopsy findings were as follows: comb and wattles, cyanotic; rigor mortis, marked; skin at the pectoral region is adherent to the breast-bone by a tenacious, yellow exudate; anterior half is pale; crop filled with grains of "palay" and oats; pericardial fluid, tinged with blood; epicardium petechiated at the coronary region; lungs, pale and bloodless; liver is dark brown in color and shows numerous petechiae; also few pin-point areas of degeneration; gall-bladder, distended with dark bile; spleen is soft; malpighian corpuscles prominent; the fat around the gizzard is reddened; duodenum, pale and contains a yellow, pasty mass; six inches from the cecum, the serosa is studded with parasitic nodules; kidneys, reddened and swollen.

On May 27, 1925, a pigeon was injected into the breast muscles with 0.5 cc of a saline suspension of a 48-hour culture of the organism. The following morning, in less than 24 hours, this pigeon died. A typical, straw-yellow, necrotic tract was found along the path of the needle. The pectoral muscles on this side were pale and infiltrated with serous fluid.

Discussion

Bipolar organisms are usually present in the soil, stagnant water, decaying plant and animal matter. They are normal saprophytes of the digestive tract and the respiratory passages. Due to certain unknown causes, besides those contributing to a lowered body resistance, these organisms become virulent, and successfully invade all the tissues of the animal. After passage through several individuals, they acquire such pathogenicity as to give rise to an enzootic or an epizootic. A gradual and sometimes a sudden subsidence of the affection, with recurrence after a certain period of time, is characteristic of hemorrhagic septicemia diseases. Thus, an epizootic of fowl cholera may apparently disappear after sanitary measures have been instituted, and break out the following year with more or less increased virulence.

In the outbreak herein discussed, bipolar organisms were recovered from the heart, liver, and spleen of fowls dead of the disease. Koch's postulates were satisfied several times, and there is no doubt that in this instance we are dealing with an epizootic of fowl cholera. On further inquiry, the disease appears to be enzootic among the fowls of the Poultry Division of the College of Agriculture, as from time to time similar outbreaks, although not as intensive as the recent outbreak, had been noticed.

Fowl plague is eliminated because filtration of the infected blood, diluted in salt solution, through a Berkefeld filter and the injection of 5 cc of the filtrate into healthy birds did not reproduce the disease. It will be noticed that the autopsy findings, whether from chickens dead in the field or from those experimentally injected, are practically identical. These lesions correspond in most respects with morbid changes described by various investigators in fowl cholera. It is, however, interesting to note that a fibrinous pericarditis was frequently observed, a lesion not often reported by other writers.

The injection of a 48-hour culture into the breast muscle of a pigeon, resulting in death within 24 hours and showing a straw-yellow, necrotic tract along the path of the needle, is characteristic of *Pasteurella avicida*, according to Hadley.

It is differentiated from fowl typhoid, because there was no leukemia present, and the cyanosis of the comb and wattles, plus the hemorrhagic duodenitis and multiple focal necroses of the liver, are diagnostic features of fowl cholera.

The biologic characters and staining qualities of the organisms studied correspond with those of *P. avicida*, the causative agent of fowl cholera.

Further studies are in progress with reference to the serological reactions of the organism and its biologic characters in the different sugar media.

Treatment

Treatment of individual fowls is not advisable. As in practically all fowl diseases, prevention is better than cure. Good sanitary conditions should be observed throughout the pens at all times, and a constant personal supervision of the flock, in order to determine at once the birds that show the least suspicion of the disease. Care should be taken in the introduction of new fowls into the flock. These should be quarantined and watched in an isolated pen for at least a month, in order to eliminate the danger of introducing any disease. The same rule should be observed in the transportation of birds from poultry shows.

In case of a severe outbreak of fowl cholera, it is advisable and will be found to be more economical to kill all sick birds at once and attempt to save the remaining as much as possible. The latter should be divided into small groups and, wherever the disease appears, that group should be disposed of. Dead fowls should be cremated or buried with lime deeply into the ground. The roosting-boards, and chicken-houses should be thoroughly cleaned and disinfected. Lime sprinkled on the floors and grounds of infected premises every so often is advisable. Drinking-troughs should be attached to the wall, in such a way that birds cannot perch on them and soil the water. Sulphuric acid, added to the drinking water at the rate of a teaspoonful to a liter of water, will minimize possible infection through the digestive tract. The infected pens should be vacated for at least two months.

The poultryman is strongly advised against many of the so-called "fowl cholera cures," as this is only a waste of time and money.

Summary

1. An epizootic of fowls, turkeys, and ducks is herein described.

2. The causative agent of this epizootic was found to be the *Pasteurella avicida*.

3. The symptoms of the disease are: (a) sudden death, (b) high fever, (c) somnolence, (d) inappetence, and (e) general weakness.

4. The morbid anatomy includes: (a) cyanosis of comb and wattles, (b) petechiae of the coronary region of the heart, (c) fibrinous pericarditis, (d) edema or hemorrhagic inflammation of the lungs, (e) liver riddled with greyish-white foci of degeneration, varying in size from a pin-point to a pin-head, (f) hemorrhagic inflammation of the duodenum.

5. Good sanitary condition of the pens at all times is strongly recommended.

6. The poultryman is strongly advised against keeping a large number of fowls together in a flock, unless provision is made for dividing the grounds and flock in case cholera or any infectious disease breaks out.

ACKNOWLEDGMENT

The writer desires to express his acknowledgment to Dr. F. M. Fronda, of the Poultry Division of the College of Agriculture, for his cooperation in submitting all dead birds for autopsy, and for some of the birds used in these experiments.

PERSONALS

Dr. J. W. Parker (K. C. V. C. '00) recently addressed the Kansas City U. S. D. A. Club on the meat inspection and stockyards inspection services of the Department of Agriculture.

Dr. L. J. Tompkins (Corn. '13), Sanitary Supervisor for the Sheffield Farms Co., Inc., of New York City, now has his headquarters at 737 Broadway, Newbergh, N. Y., where he recently purchased a new home.

Dr. Guy P. Hatchett (Ind. '22), who has been assistant state veterinarian of Tennessee for the past three years, has resigned his position to enter partnership with Dr. F. W. Morgan (McK. '06), of Chattanooga, Tenn.

Dr. H. F. Dailey (U. P. '13), chief of the veterinary staff of the Angell Memorial Hospital, of Boston, recently spoke over the radio from Station WEEI. His subject was "What the Angell Hospital Is Doing for Animals."

Dean V. A. Moore, of Cornell University, addressed the Rotary Club of Philadelphia, at the Thanksgiving Day luncheon, November 25, 1925, on the subject: "Veterinary Science as an Economic Factor." Dr. F. H. Schneider (Col. '96) acted as chairman of the meeting.

Dr. H. F. Kern (Colo. '11), who has been with the U. S. Bureau of Animal Industry since leaving the Philippine Department of Agriculture, several years ago, has accepted a position as veterinarian to the Salvador Government. The latter government is establishing a new experiment station at San Miguel, and Dr. Kern will be in charge of the veterinary work in connection with it.

ABSTRACTS

Die Aufzuchtkrankheiten des Geflügels und ihre Bekämpfungunter besonderer Berücksichtigung der tuberkulose (The Diseases of Growing Birds and their Prevention, with Special Reference to Tuberculosis). M. Lerche. Deutsche Tierärztliche Wochenschrift, Hannover, xxxiii (1925), pp. 840-843.

The author discusses the diseases that have been found to affect young birds. He urges the use of the tuberculin test in combating tuberculosis in poultry, and states that he had conducted experiments on the problem of no-lesion reactors, finding that birds may give a positive tuberculin reaction as early as seven to eleven days after artificial infection. No macroscopic lesions could be found in these early stages of the infection, but microscopic lesions could be demonstrated.

Attempts were made to immunize birds with tubercle bacilli from cold-blooded animals. The results were favorable, but he advises against the promiscuous employment of this method of prevention, since it brings about a greater danger of spread by immune carriers.

H. J. S.

The Effect of Feeding Bacillus Acidophilus, Lactose, Dry Skim Milk, or Whole Milk on the Hydrogen Ion Concentration of the Contents of the Ceca of Chicks. J. R. Beach. Hilgardia, i (1925), 8, pp. 146-166.

The experiments here reported were undertaken to determine in what manner, if any, hydrogen ion concentration of the cecal contents of chicks would be influenced by feeding them with milk or certain milk products, and the relation of changes, if any were noted, to the control of coccidiosis.

The author summarizes his findings and concludes as follows:

Feeding milk cultures of *B. acidophilus* to chickens resulted in the implantation of *B. acidophilus* in the ceca. In some instances, nearly 100 per cent of the bacteria present in smears of the cecal contents, stained by Gram's method, were of the acidophilus type. The implantation of *B. acidophilus* in the ceca of chickens, however, did not change the pH value of the cecal contents.

The droppings of chickens originating in the ceca are voided separately, and can be differentiated from the droppings from other portions of the intestinal tract. It is possible, therefore, to study changes in the cecal contents of the same chicken that occur from day to day.

The pH of the cecal contents of chickens was changed from the normal range of 6.0 to 7.4 to a range of 4.4 to 5.6 by feeding sufficient amounts of whole sweet milk, milk cultures of *B. acidophilus*, milk cultures of *B. acidophilus* plus lactose, lactose alone, or dry skim milk.

Since lactose is the only ingredient common to all of the milk products used, the change in hydrogen ion concentration of the cecal contents produced by feeding milk or a milk product would appear to be due to the lactose it contains.

The change in the hydrogen ion concentration of cecal contents from a single feeding of a milk product, occurred within two to two and one-half hours after the feeding, and returned to normal within eight to twenty-four hours after the feeding. The rapidity of development and the short duration of the change in hydrogen ion concentration indicate that these are not the result of modification of the flora of the intestinal tract.

An abnormal degree of acidity in the ceca was constantly maintained by the individual administration, to chickens, of one or two grams of lactose twice each day at an interval of about eight hours, or by the continuous feeding of mash mixtures containing 20 per cent of lactose. The feeding of mash containing 40 per cent dry skim milk would also provide approximately 20 per cent of lactose in the mash and should, therefore, accomplish the same result.

THE INFLUENCE OF FEEDING LACTOSE OR DRY SKIM MILK ON ARTIFICIAL INFECTION OF CHICKS WITH EIMERIA AVIUM. J. R. Beach and D. E. Davis. Hilgardia, i (1925), 8, pp. 169-181.

In this paper, the authors report the results of five attempts to combat artificially produced coccidiosis in chicks by feeding them with sufficient lactose or dry skim milk, to change the pH of the ceca from the normal range of 6.0-7.4 to a range of 4.4-5.6. The authors believed that, by this means, an environment unfavorable or even destructive to the tissue-invading stages of the parasite might be established.

As stated by the authors, in their summary and conclusions, "The results of the series of five experiments were uniform in demonstrating that chicks were afforded a considerable degree of protection against coccidial infection, when a sufficient amount of lactose or dry skim milk was added to their diet. In the trials, carried out under laboratory conditions, this was accomplished equally well by the individual administration of two 1-gram doses of lactose to each bird daily, at an interval of about eight hours, or by feeding chicks continuously with mash containing 20 per cent lactose or 40 per cent dry skim milk. In the trials carried out under field conditions, however, the results obtained from the use of skim-milk powder were superior to those obtained from the use of lactose. This was due, at least in part, to the fact that the chicks did not relish the mash mixture containing lactose and, therefore, consumed less of this mash than of that containing dry skim milk. The relatively greater increase in weight of the chicks fed on dry skim milk indicated that the superior food value of this material was also, at least in part, responsible for the benefit derived from its use.

"The results of these experiments confirm those described in the preceding paper, in showing that when sufficient lactose or dry skim milk is fed to chickens, the hydrogen ion concentration of the cecal contents can be kept within a range of 4.4 to 5.6. It is thought that this degree of acidity may be sufficient to injure or destroy the sporozoite or merozoite forms of *Eimeria avium*, and that serious harm from the infection is thereby prevented. However, both merozoites and öocysts were found in the droppings of birds inoculated with sporulated öocysts and treated with lactose or dry skim milk, even though the birds showed no visible signs of sickness after the inoculation. This is evidence that at least a part of the sporozoites released from the sporocysts were unharmed, and invaded the cells of the cecal mucosa where both the sexual and asexual cycles of development were completed. A possible explanation of this is that the dose of sporulated öocysts given was too large to be entirely overcome and, therefore, a portion of the sporozoites escaped. Another possible explanation is that the acidity in the ceca was more destructive to the merozoites than to the sporozoites. In such a case, the invasion of the epithelial cells by the sporozoites and the completion of the developmental cycles within the cells would be unhindered. The merozoites, how-

ever, upon emergence from the epithelial cells into the acid cecal contents would be destroyed, and further development of disease arrested. On this basis, the appearance of blood in the droppings, and death on the fifth and sixth days after inoculation, of some of the birds which were fed lactose or skim-milk powder, could be ascribed to the tissue damage resulting from the initial invasion with sporozoites. The destruction of the merozoites, however, prevented further development of disease in the birds which were not fatally injured by the sporozoite invasion.

"This explanation would not apply to the failure of lactose feeding, in the last two coccidiosis-control trials, to afford the chicks as high a degree of protection against coccidial infection as was given by dry skim milk. This, as previously pointed out, was probably due in part to the difference in amount of consumption of the two mash mixtures by the chicks (12.8 per cent less of lactose), and also in part to the superior food value of the skim-milk powder.

"The fact that feeding chickens mash containing 40 per cent dry skim milk not only protected them against coccidial infection, but also stimulated rapid growth, indicated that this would be a valuable practice in the prevention and control of outbreaks of the disease on poultry farms."

GEFLÜGELTUBERKULOSE BEI SÄUGERTIEREN (Avian Tuberculosis in Domestic Animals). N. Plum, Veterinary College, Copenhagen, 1925.

This publication contains an extensive review of the literature. A large number of references are given and in many instances a rather complete abstract. For example, concerning the occurrence of avian tuberculosis in man, twenty references and summaries are noted. As a result of this review, the author states that the subcutaneous injection of reasonable quantities of avian tubercle bacteria, a local reaction is set up or this reaction may extend to the local lymph-glands. In the smaller experiment animals, uniform results can be obtained only if very large quantities of the virus in proportion to body weight are employed. Intravenous and intraperitoneal injections of avian tubercle bacteria, in large amounts, produce tuberculosis in some animals and in others produce no effect. In all animals, even those which are quite non-susceptible, large doses of virulent material may be

followed by death, due to the toxic action of the injected dose. Death may be preceded by cachectic symptoms which may be associated with atrophy of certain of the organs. With the injection of large quantities of virulent material in any animal, one might induce death due to the Yersin type of the disease. Young animals may become infected by feeding sufficient quantities of avian tubercle bacteria of known virulence. Older animals are much more immune to this method of infection.

Plum's own work was confined largely to the study of bovine abortion, due to the avian tubercle bacillus. Nine cases are described. The first five were afterbirths from abortions. From each of these, avian tubercle bacteria were isolated.

In the sixth case the material studied was uterine exudate. This was injected into hens, rabbits and guinea pigs. The rabbits and hens developed tuberculosis. The guinea pigs were not infected. Case seven consisted of three calves, only one of which was found tuberculous. Avian tubercle bacteria were demonstrated by inoculation, in the mesenteric lymph-glands. Case eight was a steer from which avian tubercle bacteria were demonstrated, by animal inoculation, in the retropharyngeal lymph-gland. Case nine was also a calf which reacted to avian tuberculin and avian tubercle bacteria were demonstrated in the retropharyngeal lymph-glands.

Some of the animals affected naturally with avian tuberculosis were tested and most of them reacted to avian tuberculin, and not to bovine tuberculin. The whole herd was tested in some instances and additional reactors found. A few cases reponded to both avian and bovine tuberculin. The intradermal test seemed to give better results than the subcutaneous. A large number of inoculations of avian tubercle bacteria were made into various species of animals. These were subsequently tested with quite variable results.

The work here reported is very interesting and valuable and should be studied by all interested in the problem of avian tuberculosis.

C. P. F.

Harvard University is making plans for a graduate school of agriculture.

The Minnesota Cooperative Creameries sold 26 carloads of butter on the Pacific Coast in July and August of 1925.

ARMY VETERINARY SERVICE

CHANGES RELATIVE TO VETERINARY OFFICERS

Regular Army

First Lieutenant James L. Barringer, V. C., promoted to rank of Captain, December 16, 1925.

Reserve Corps

New Acceptances

Major:
Ticehurst, Harry.....County Road, Tenafly, N. J.
First Lieutenant:
Basinger, Harry Phillip.....120 N. Kline St., Aberdeen, S. D.
Second Lieutenant:
Bassett, Dell Clare.....61 Marvin Avenue, Hempstead, L. I., N. Y.
Jones, James Verne.....2219 Harrison Ave., Cincinnati, Ohio.
Kinney, Wm. Graham .....1101 West Lake Avenue North, Seattle, Wash.

Promotions:

Second Lieut. Thomas Boyd Jacobs, Newberry, S. C. promoted to grade of First Lieutenant.

Separations.

First Lieut. Edward H. Lenheim, Burlingame, Kansas, declined reappointment.

HORSES PULLED THEM OUT

In the *Field Artillery Journal*, Captain John H. Fye, in reporting upon the 1925 field training of the 119th Field Artillery of the Michigan National Guard, says:

"The regiment arrived at Alger in a pouring rain which continued throughout the afternoon and night. Naturally everything was drenched by next morning. The camp site was a good one but the clay soil became so sticky that the march of the regiment was delayed while the teams pulled the trucks and motorized rolling kitchens to the highway. If the march had begun without getting the motorized consolidated kitchens on the road, it is very doubtful if the regiment would have eaten on the remainder of the hike. Once more the horse fans were heard to say, 'The old horse triumphs again over the motor'—The scene reminded one of the muddy days in sunny France."

ARMY HORSES IN INDIA

The system of numbering the horses in India on a general roll is being abolished and in future the horses will be given unit numbers and be branded with them. A continuous and detailed record of the history of each animal will be kept from the day of purchase to the end of service, and the veterinary record kept in the units will give full detiils of all ailments and treatment.

—*The Veterinary Record.*

The Cuban National Veterinary Association has arranged for a diploma of honor to be awarded to the member of the Veterinary Corps of the Cuban National Army attaining the highest standing.

COMMUNICATIONS

THE OLD ARMY VETERINARIANS

To the Editor:

My good friend of many years, Dr. Jas. A. Waugh, misinterprets my remarks in discussing Dean Wegner's paper. What I did say was, "Most of you can remember when conditions were such that well-trained veterinarians were not attracted by the army service." I certainly did not mean to infer that there were not some splendid veterinarians in the army, in spite of the objectionable conditions—and these men performed an excellent service in building up the army veterinary service to what it is now. They deserve much credit. In addition to the names mentioned by Dr. Waugh, there are many still on the active list of the army veterinary service. I think Dr. Waugh himself gave his excellent services for a time, but resigned, as did the late Dr. Benj. D. Pierce, and we infer that they left the army because of the objectionable conditions. Dr. Waugh well knows the trying conditions under which veterinarians served in the old days, and to which I referred. First, the veterinarian had *no rank*, he was neither an officer nor an enlisted man. The pay and allowances were small. There was no retirement with pay, nor was there promotion.

Dr. Waugh infers that I do not use correct English. I humbly confess that I am not perfect in this, nor in many other things.

N. S. Mayo.

Chicago, Ill., January 12, 1926.

CORRECTION

Dr. P. T. Engard calls attention to the fact that, in his communication, "What Is the Trouble?" published in the January issue of the Journal, he intended to refer to a number of state experiment stations supplying cuts for the purpose mentioned, and not to the Ohio Agricultural Experiment Station.

Agricultura y Zootecnia, an excellent agricultural and live stock paper published in Havana, speaks very highly of the efficient organization and personnel of the Cuban Army Veterinary Corps, of which Lieutenant Colonel Luis A. Beltran (U. P. '08) is at the head. The Corps is composed of two lieutenant colonels, two majors, nine captains and thirteen first lieutenants.

ASSOCIATION MEETINGS

NORTH CENTRAL IOWA VETERINARY MEDICAL ASSOCIATION

The fall meeting of the North Central Iowa Veterinary Medical Association was held at Fort Dodge, December 10, 1925. The meeting was called to order by the President, Dr. E. R. Truax, of Sac City.

We were favored with an especially fine program, among the outstanding papers being one given by Dr. Charles Murray, Veterinary Division, Iowa State College, on "Diseases of Poultry." This was an illustrated lecture and an opportunity was had to examine a number of fowls postmortem. Dr. Murray covered in detail the commonly encountered poultry diseases.

Dr. A. T. Kinsley, of Kansas City, Mo., gave a very interesting and instructive paper on "Diseases of Swine." Following Dr. Kinsley's paper he had charge of a "question box." The members submitted written questions concerning problems in practice, and these were discussed in open meeting. It was felt that much good was derived from the free discussion of Dr. Kinsley's very practical paper, and the large number of questions submitted.

The meeting was then adjourned until evening, when a dinner and dance, given by the Fort Dodge Serum Company, were enjoyed by the members, their wives and friends.

H. J. SHORE, *Secretary.*

UNIVERSITY OF MISSOURI FIRST SPECIAL COURSE FOR VETERINARIANS

The first special course for graduate veterinarians, given by the University of Missouri cooperating with the Missouri Veterinary Medical Association, was convened at the Veterinary Building of the University of Missouri, Columbia, December 28, 1925, and continued for four days.

The enrollment of over sixty graduate veterinarians was noted with much pleasure on the part of those who had labored to make the course a success. One instructor stated that it was an outstanding success in that the attendants were on time and stayed throughout each lecture or demonstration.

Graduate Veterinarians Attending Special Course given at the Vet. Dept
College of Agriculture, University of Missouri.
Missouri Veterinary Medical Association Co-operating Dec. 28-31-1925.

FIRST SPECIAL COURSE FOR GRADUATE VETERINARIANS, UNIVERSITY OF MISSOURI, COLUMBIA, MO., DEC. 28-31, 1925.

(1) C. H. Honeywell, Slater; (2) P. C. Denney, Mountain Grove; (3) E. L. Dudgeon, Platte City; (4) Ray Matkin, Rockport; (5) E. D. Criswell, King City; (6) (6) L. L. Beck, Pattonsburg; (7) P. C. Lahs, Independence; (8) J. S. McDaniel, Lexington; (9) H. A. Wilson, Jefferson City; (10) Stanley Smith, Columbia; (11) D. F. Luckey, Columbia; (12) H. W. Young, Columbia; (13) H. A. Presler, Hannibal; (14) H. C. Ward, Fulton; (15) R. F. Buell, Vandalia; (16) Ralph Graham, Jefferson City; (17) A. J. Durant, Columbia; (18) E. L. Young, Grandview; (19) F. H. Suits, Odessa; (20) W. J. Stone, Joplin; (21) G. W. McIntyre, Mexico; (22) Harold Newman, Columbia; (23) A. Goodlive, Marshall; (24) J. C. King, Providence, Ky.; (25) .....; (26) C. R. Kinnison, Chillicothe; (27) G. H. Elliott, Palmyra; (28) S. G. Dreppard, West Plains; (29) O. T. Murphy, Kahoka; (30) T. J. Eagle, Bethany; (31) E. S. Harrison, Burlington Junction; (32) E O. Lueking, Desoto; (33) August Luh, Victoria; (34) W. T. Duncan, Springfield; (35) H. L. Bussong, Belton; (36) E. T. Hallman, East Lansing, Mich.; (37) C. D. Lueckert, Blackwater; (38) C. A. Schulz, Independence; (39) J. M. Burns, New Hampton; (40) W. H. Thomas, St. Joseph; (41) B. M. Miller, California; (42) Andrew Uren, Columbia; (43) D. B. Morgan, Neosho; (44) Roy Morgan, Skidmore; (45) W. B. Welch, Marshall; (46) R. L. Allen, Windsor; (47) Horace Bradley, Clayton; (48) C. D. Meredith, Joplin; (49) O. S. Crisler, Columbia; (50) John W. Adams, Philadelphia, Pa.; (51) F. R. Whipple, Peoria, Ill.; (52) H. M. McConnell, Independence; (53) F. C. Cater, Sedalia; (54) J. W. Connaway, Columbia; (56) Ray Mier, Smithville; (57) M. Gregory, Jefferson City.

Dr. John W. Adams, president of the American Veterinary Medical Association, was present for two days and gave freely of his skill in operating and instruction.

Dr. R. R. Dykstra, dean of the Veterinary Division, Kansas State Agricultural College, gave teat surgery and other surgical lectures of much interest. Dr. E. T. Hallman, of Michigan State College, covered sterility, with both lectures and demonstrations. Dr. Frederick R. Whipple, of Peoria, Ill., had charge of the small animal division, demonstrating endoscopic instruments and performing the "barkless dog" operation (ventriculochordectomy). Dr. Sivert Eriksen, pathologist, Missouri Poultry Experiment Station, Mountain Grove, had the poultry division. He lectured and held a clinic in which each veterinarian had a subject to work upon.

Dr. E. M. Nighbert, Zoological Division, Bureau of Animal Industry, gave diseases of sheep, with special emphasis on the parasitic diseases.

Of those from the staff of the University who gave material aid to the program were: Dean F. B. Mumford, Dr. M. P. Ravenel, Professor of Bacteriology, and Dr. J. W. Connaway. Dr. A. J. Durant, Dr. A. Uren and Mr. H. G. Newman, of the staff of Dr. Connaway, also assisted.

The Animal Husbandry Division gave some good practical demonstrations of their efforts with the University's pure-bred herds, Professors E. A. Trowbridge, A. C. Ragsdale, L. A. Weaver, and D. W. Chittenden participating, leaving much food for thought.

From the Poultry Department, Professors E. W. Henderson and H. L. Schrader gave talks and demonstrations on culling, feeding and housing, which were highly instructive and appreciated.

The only unsatisfactory thing about the course was, as so aptly expressed by one veterinarian present: "It was only for four days, when it should have been for two weeks."

The splendid cooperation of the entire staff of instructors, in each department of the University, was one of the pleasant things encountered and deeply impressed itself on the minds of those in attendance, many of whom charged me to convey their heartfelt appreciation of the treatment received by them. That this will be an annual event in the future we have no occasion to doubt.

FRED C. CATER, *Secretary.*

Independence; (53) F. C. Cater, Sedalia; (54) J. W. Connaway, Columbia; (56) Ray Mier, Smithville; (57) M. Gregory, Jefferson City.

MISCELLANEOUS

ANOTHER OPPORTUNITY FOR PRACTITIONERS IN PENNSYLVANIA

In addition to culling and certifying flocks, the Pennsylvania Bureau of Markets, in cooperation with the Pennsylvania Bureau of Animal Industry, will accredit flocks for bacillary white diarrhea for those owners who desire same.

The Bureau of Animal Industry has supervision over the collecting and examining of the blood. The plan in Pennsylvania is, briefly, as follows:

The Bureau of Markets makes the preliminary arrangements. A date satisfactory to the owner, the local practicing veterinarian and the Bureau of Animal Industry is set for collecting the blood. A Bureau of Animal Industry veterinarian, specially trained in poultry diseases, is present for the purpose of assisting the local practicing veterinarian in collecting the blood and packing for shipment to the Bureau of Animal Industry laboratory at Philadelphia, and to get him otherwise properly started in the work. Unless the blood is in good condition upon arrival at the laboratory, a dependable examination can not be made. Great care, then, must be exercised in the collecting, packing and mailing of the blood to the laboratory.

Poultrymen and other laymen have been in the habit of collecting blood themselves. White diarrhea work is disease work and the collecting of the blood as well as giving advice regarding the disease should be done by veterinarians. Since it has been necessary in most cases to have a Bureau representative present to assist the local practicing veterinarian in collecting the blood, the poultrymen would not understand why they should have to pay the local practicing veterinarian for his work. So it was thought advisable for the State to collect a fee from the owner and pay the local veterinarian for his services. This plan will be followed until the work is established and the poultrymen are accustomed to calling upon the practitioners to perform this kind of service.

This, the first year, resulted in the collecting and examining in Pennsylvania of blood specimens from twenty (20) flocks consisting of 12,697 chickens. Indications are that there will be a greater demand for this kind of veterinary service next year.

Accrediting poultry flocks for bacillary white diarrhea opens up a new field for veterinary practice. This work serves a two-fold purpose. It affords the practitioners splendid opportunity to demonstrate to poultrymen their ability to render veterinary service and, second, to gain additional, valuable, poultry-husbandry information and poultry-disease experience.

In this, as in all poultry- and animal-disease prevention and control field-work, the Pennsylvania Bureau of Animal Industry operates through and in cooperation with the practicing veterinarians of the State.

HOG CHOLERA LOSSES REDUCED

At no time since records of losses from hog cholera have been kept has the death rate from this disease been so low as during the fiscal year 1925. The disease was at a low ebb throughout the year, and the losses were light compared with those of any previous year. This year only 30.88 hogs out of every 1,000 on farms in the United States died of hog cholera, whereas in 1897 the loss was 130 and in 1914 it was 106 per 1,000. A disturbing factor, however, is the fact that many of the swine herds on farms have been left susceptible to hog cholera. Since the disease has not been so prevalent as usual, farmers have failed to have their hogs immunized, and were the disease to begin spreading again some heavy losses would probably occur before outbreaks could be checked. (Report of Chief of Bureau of Animal Industry, 1925.)

STEEL MEMORIAL MEDAL AWARD

Accoring to an announcement in *The Veterinary Record*, the Council of the Royal College of Veterinary Surgeons has awarded the Steel Medal for veterinary research to Professor S. H. Gaiger, who was recently appointed to the chair of the Care of Animals—Causation and Prevention of Disease, in the University of Liverpool. It is nearly twenty years since this medal was awarded by the Council to a member of the Council. The award is made once in every three years.

42 PIGS IN SIX MONTHS RECORD OF THIS SOW

A Kansas farmer owns a sow worth her weight in gold. Last spring the sow farrowed 24 pigs. Then the latter part of August she added 18 more, making 42 pigs in less than six months.

NECROLOGY

VEARL A. HEATER

Dr. Vearl A. Heater, of Fort Dodge, Iowa, died December 23, 1925, at a Nevada, Iowa, sanitarium, following an acute complication of a stomach trouble from which he had suffered for several months.

Born in Portland, Ohio, July 26, 1894, Dr. Heater, affectionately known to friends and fellow professionals as "Chick" Heater, was only thirty-one years of age at the time of his death, and was one of the most popular of the Midwest's younger veterinarians. He is survived by his widow, nee Ethel Baughman, daughter of Dr. and Mrs. D. E. Baughman, of Fort Dodge, and a three-year-old son, Robert.

During his collegiate years at Iowa State College, from which institution he received his veterinary degree in 1919, Dr. Heater was prominent in athletic and fraternity circles. He was chosen as an all-Missouri Valley Conference football player and received many other outstanding athletic honors. His interest and participation in athletics continued after graduation. He was a fine horseman and took keen interest in polo and other equestrian sports.

Although a young man, Dr. Heater was, at the time of his death, general superintendent of the Fort Dodge Serum Company, Fort Dodge, Iowa. He joined the A. V. M. A. in 1923. He was a member of the Kiwanis Club, Masonic Lodge, American Legion and Phi Gamma Delta fraternity.

Dr. Heater's death is not only a great shock and misfortune to his devoted family and other dear ones, but to the veterinary profession as well.

S. L. B.

MILTON R. TRUMBOWER

Dr. Milton R. Trumbower died at his home, in Monett, Mo., January 4, 1926, in his seventy-eighth year.

A native of Pennsylvania, Dr. Trumbower first studied human medicine, but later decided to take up veterinary medicine, and was graduated from the American Veterinary College, in New York City, in 1886. He practiced for many years in Sterling, Ill., removing to Missouri about twenty years ago. After finishing

a medical course in St. Louis, Dr. Trumbower was admitted to practice. He located in Monett, and very soon built up a very large practice there.

During the time that Dr. Trumbower was located in Illinois, he was state veterinarian (1893-1897). This was during the administration of Governor Altgeld, and it was during this period that the first tuberculin tests in Illinois were made, under Dr. Trumbower's direction, by Dr. Harry G. Hoover, of Sterling.

Dr. Trumbower joined the A. V. M. A. in 1886, and took an active part in the Association until he took up human medicine. He was also a member of the American Medical Association. For several years, he was Professor of Cattle Pathology and Meat and Milk Inspection in the Chicago Veterinary College. He also held the positions of Veterinary Inspector in the Bureau of Animal Industry and Meat Inspector for the city of Chicago.

EDWARD C. COLEMAN

Dr. Edward C. Coleman, of Detroit, Mich., died December 17, 1925, following an operation, the seventh to which he was obliged to submit during recent years. Dr. Coleman was a graduate of McKillip Veterinary College, class of 1920, and conducted an exclusively small animal practice in Detroit.

PATRICK E. QUINN

Dr. Patrick E. Quinn died at Harrisburg, Pa., January 17, 1926, following a short illness due to pneumonia.

Born at Indianapolis, Ind., March 29, 1872, Dr. Quinn entered the service of the Bureau of Animal Industry as a tagger, December 11, 1895. Five years later he decided to take up the study of veterinary medicine and entered the Cincinnati Veterinary College, from which institution he was graduated in 1903. After his graduation he was appointed assistant inspector in the B. A. I. and served in various capacities, principally in the Meat Inspection Division, until March, 1918, when he was transferred to tuberculosis eradication work and assigned to Washington, D. C. On June 17, 1918, he was transferred to Harrisburg, Pa., as inspector in charge of tuberculosis eradication work in Pennsylvania, which position he held up to the time of his death.

Dr. Quinn joined the A. V. M. A. in 1915.

In cooperation with the state live stock sanitary officials of Pennsylvania, Dr. Quinn has watched the tuberculosis eradication work expand from a small beginning to its present large volume. With the officials of the Keystone State he has been instrumental in founding the policy now in effect and in placing tuberculosis eradication work on a solid foundation. He was a capable and conscientious official and through his pleasing personality he had endeared himself to his many friends and associates.

Dr. Quinn is survived by his widow and four children.

MRS. JAMES LAW

Mrs. James Law, widow of the late Dr. James Law, died at her home in Ithaca, N. Y., December 12, 1925, in her 88th year. She had been in fair health for a woman of her advanced age and the end came while she slept.

J. NORMAN BROWN

Dr. J. Norman Brown, of Pittsfield, Maine, died at his home, January 21, 1926. He had been in failing health for a long period.

Born in Guilford, Me., October 27, 1887, he was graduated from Dexter (Me.) High School in 1908 and from the Ontario Veternary College in 1912. He located in Pittsfield immediately and was a successful practitioner.

Dr. Brown is survived by his widow, his mother and a sister.

C. A. WASHBURN

Dr. C. A. Washburn, aged 67 years, for more than thirty years a veterinary practitioner of Mayfield and Groves counties, Kentucky, passed away, November 13, 1925, at his home in Mayfield, Ky.

V. N. ONISSIMENKO

An Associated Press dispatch from Honolulu, dated January 14, 1926, reports the death of Dr. Vassily Nicholaevich Onissimenko, deputy territorial veterinarian and bacteriologist for the island of Maui, due to anthrax. We hope to have authentic information for publication in the next issue of the JOURNAL.

Dr. William Titus, of Grandview, Ind., died November 9, 1925, at the age of 74 years.

MARRIAGES

Dr. J. A. Holzman, of Dallas, Texas, to Miss Lilly Platte, of Waco, Texas, November 5, 1925.

Dr. George Hewett (Corn. '16), of Albany, N. Y., to Miss Muriel Aislie, of Albany, N. Y., November 26, 1925.

Dr. David S. White (O. S. U. '90), of Columbus, Ohio, to Miss Mabel Elizabeth Moran, of Columbus, Ohio, December 21, 1925, at Covington, Ky.

BIRTHS

To Dr. and Mrs. L. H. Bennett, of Monroe, La., a son, Leslie Herman.

To Dr. and Mrs. A. C. Yow, of Henderson, N. C., a son, Alexander Jensen.

To Dr. and Mrs. J. H. Copenhaver, of Ralston, Nebr., a daughter, Elaine, November 17, 1925.

To Dr. and Mrs. J. M. Floyd, of Lockney, Texas, a son, Shirlie Rae, November 28, 1925.

To Dr. and Mrs. J. R. Fuller, of Walla Walla, Wash., a son, Jay Robert, Dec. 22, 1925.

To Dr. and Mrs. K. H. Gubser, of Adel, Iowa, a son, Edward Alfred, Dec. 31, 1925.

To Dr. and Mrs. F. E. Lentz, of Philadelphia, Pa., a son, Frank E. Jr., January 9, 1926.

PERSONALS

Dr. Peter F. Runyon (U. P. '12) was recently elected Mayor of Freehold, N. J.

Dr. C. H. Taylor (Corn. '05) has removed from DeKalb, Ill., to Cortland, N. Y.

Dr. William H. Dohm (Corn. '23), of Ossining, N. Y., has removed to Hollywood, Fla.

Dr. G. A. Unbewust (Wash. '16) has removed from Lebanon, Ore., to Harrington, Wash.

Dr. Claude D. Evans (McK. '16), of Kane, Pa., has been elected a member of the Borough Council.

Dr. J. J. Law (Gr. Rap. '06), who recently practiced in Lansing, Mich., has removed to Caro, Mich.

Dr. R. M. Platt (K. S. A. C. '10) has changed his address from Coldwater, Kans., to Hoopup, Colo.

Dr. J. O. McCoy (Ont. '25), formerly of Success, Sask., is now associated with Dr. Willis L. Brenton (Ont. '03), of Detroit, Mich.

Dr. C. C. Stevens (Ont. '02), of Port Huron, Mich., has been re-elected City Veterinarian for a two-year term, dating from December 1, 1925.

Dr. A. H. Quin, Jr., (Chi. '20) is back with the Fort Dodge Serum Co., and has his headquarters at 636 Des Moines St., Des Moines, Iowa.

Dr. R. M. Staley (U. P. '06) has resigned his connection with the H. K. Mulford Company, of Philadelphia, and is now engaged in general practice at Narberth, Pa.

Dr. G. Gus E. Vaselius (Corn. '22), of Spencer, N. Y., has been reappointed County Veterinarian for Chemung County (N. Y.) in connection with tuberculosis eradication work.

Dr. L. A. Klein (U. P. '97), Dean of the School of Veterinary Medicine of the University of Pennsylvania, has returned from his sojourn in Texas, considerably improved in health.

Dr. B. E. Miller (Gr. Rap. '12), who has practiced at Nashville, Mich., for the past thirteen years, has removed to Ann Arbor, Mich., where he will engage in small animal practice.

Dr. Charles B. Cain (Corn. '23), of Little Springs, Miss., has accepted the position of Associate Professor of Veterinary Science at the Agricultural and Mechanical College of Mississippi.

Dr. W. E. Frink (Corn. '07), of Los Angeles, Calif., was among those who attended the recent Cornell Conference. He also visited relatives in New York State in connection with his stay in Ithaca.

Dr. S. J. Walkley (McK. '07) has sold out his real estate, loan and insurance business in Milwaukee and has been reinstated in the Bureau of Animal Industry. He is at present engaged in meat inspection work in Chicago.

Dr. John F. Bullard (Corn. '22) has resigned his position in the Department of Surgery and Medicine, Kansas State Agricultural College, to take up a new position at the Kentucky Agricultural Experiment Station, February 1.

Dr. W. A. Hilliard (McGill '97), of the Health of Animals Branch, Canada Department of Agriculture, has been transferred from Winnipeg to Emerson, Manitoba, and stationed there as Boundary Inspector, succeeding Dr. B. A. Bescoby (Ont. '10), resigned.

Dr. Waldo H. Brownell (Amer. '89), who has been located at Brockton, Mass., for a good many years, has gone to California. His new address is 1353 N. Norton Ave., Glendale. Dr. Brownell has been a reader of the JOURNAL for a continuous period of forty years.

Dr. C. M. Carpenter (Corn. '17) read a paper entitled "A Report of Two Cases of Undulant Fever in Man Due to *Brucella Abortus*," before the recent meeting of the American Society of Bacteriologists, in Madison, Wis. Dr. E. L. Brunett (Corn. '23) was also in attendance.

Dr. F. U. Fernsler (U. P. '02), of Lebanon, Pa., underwent an operation for appendicitis at the University of Pennsylvania Hospital, in Philadelphia, on January 7, 1926. The latest available report was to the effect that he was out of danger and was making a satisfactory recovery.

Dr. R. R. Birch (Corn. '12), professor of veterinary research at Cornell University, will sail for Europe, March 15, under the auspices of the International Education Board, to study diseases of cattle and swine. He will visit Copenhagen, Budapest and London, and will conduct special research work in each place. He will return to Ithaca in the fall.

Dr. E. L. Stubbs (U. P. '13) has been appointed Director of Laboratories of the Pennsylvania Bureau of Animal Industry to succeed Dr. Miller F. Barnes, resigned. Dr. Stubbs has been attached to the Laboratory and Experimental Farm branch of the State work for over twelve years, and, in addition to his studies on transmissible diseases of swine, Dr. Stubbs has specialized in poultry diseases. His work on fowl pest has attracted particular attention.